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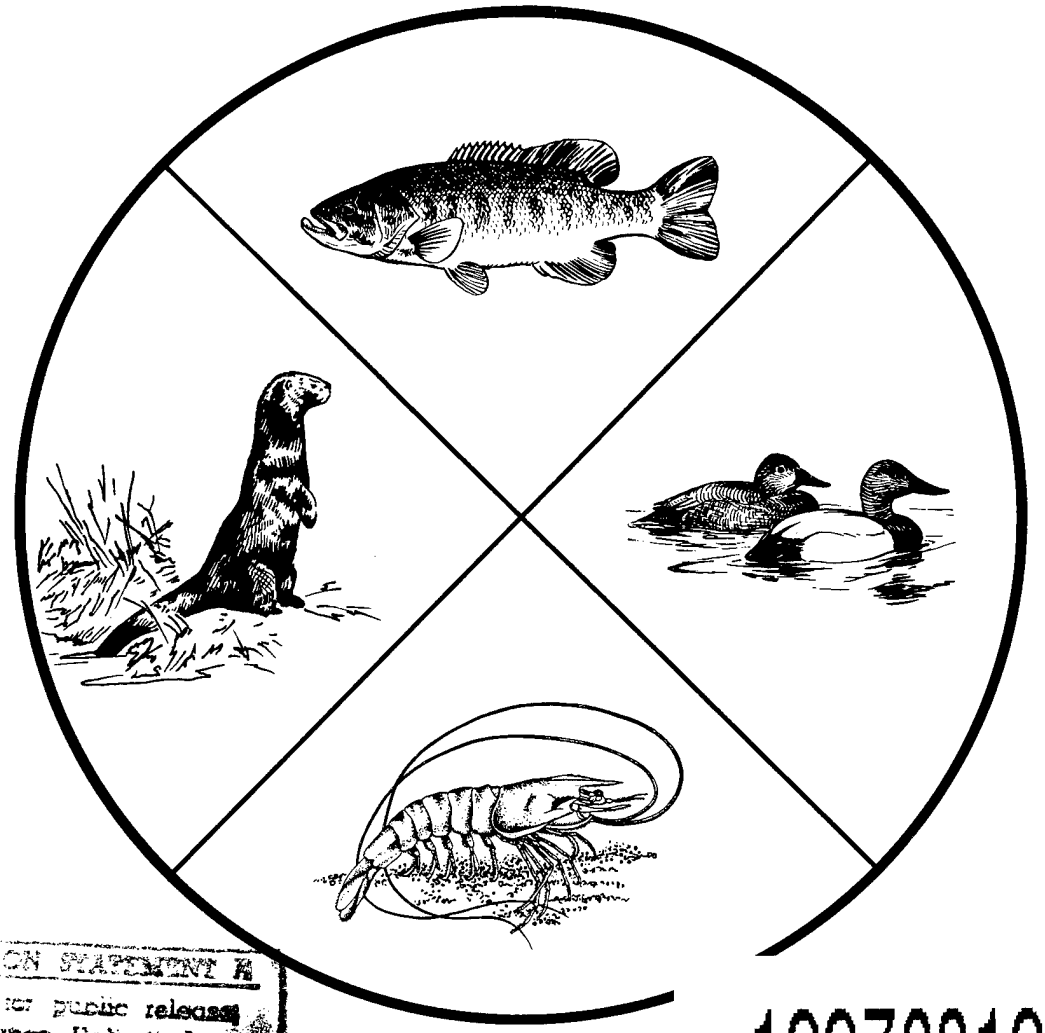
PENTACHLOROPHENOL HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW



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Contaminant Hazard Reviews
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PENTACHLOROPHENOL HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by

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SUMMARY

Pentachlorophenol (PCP) is a synthetic organochlorine compound that was first manufactured commercially in 1936 and is now used primarily as a wood preservative and secondarily as a herbicide, insecticide, fungicide, molluscicide, and bactericide. Current global production of PCP is estimated to be 50 million kg annually. Widespread use of PCP has resulted in the detection of residues in air, rain, snow, groundwater, surface water, drinking water, fish, and aquatic invertebrates, as well as in human urine, blood, and milk. Pentachlorophenol may be incorporated into animal tissues through inhalation, diet, or contact; its toxic action results from its ability to interfere with the production of high energy phosphate compounds essential for cell respiration. Pentachlorophenol has caused numerous occupational illnesses and deaths, and has had significant adverse effects on domestic animals. It is fetotoxic and teratogenic, but evidence for mutagenicity and carcinogenicity is incomplete or negative. Commercial PCP preparations often contain variable amounts of toxic impurities--including chlorophenols, hexachlorobenzene, phenoxyphenols, dioxins, and dibenzofurans--that contribute to its toxicity. Pentachlorophenol is rapidly accumulated and rapidly excreted, and has little tendency to persist in living organisms; it is readily degraded in the environment by chemical, microbiological, and photochemical processes.

In sensitive aquatic species, PCP adversely affected growth, survival, and reproduction at media concentrations of 8 to 80 ug PCP/l in algae and higher plants, at 3 to 100 ug/l in invertebrates, and <1 to 68 ug/l in fish. In birds, PCP was fatal at 380 to 580 mg/kg body weight (BW) in oral doses, >3,580 mg/kg in the diet, and >285 mg/kg in contaminated nesting materials (i.e., wood shavings). Residues >11 mg PCP/kg fresh weight in bird tissues were associated with acute toxicosis. Adverse sublethal effects in birds were noted at dietary levels as low as 1 mg/kg ration. In small laboratory mammals and domestic livestock, acute oral LD-50's ranged from 27 to 300 mg/kg BW. Tissue residues in mammals were elevated at PCP doses as low as 0.05 mg/kg BW, and at air levels >0.1 mg/m^3. Histopathology, reproductive impairment, growth retardation, and other effects were evident in sensitive mammals at PCP concentrations of 0.2 to 1.25 mg/kg BW, and at dietary levels >30 mg/kg ration.

Pentachlorophenol is an undesirable pollutant whose use patterns should be carefully regulated to avoid contamination of soil, water, and food.

Recommendations for protection of sensitive fishery and wildlife resources follow; however, it is emphasized that some of these recommendations are markedly lower than those proposed by regulatory agencies. For protection of aquatic life, it is recommended that the PCP water concentration not exceed 3.2 ug/l; but even at this level certain species of fishes and oysters accumulate enough of the toxicant to retard their growth. In birds, dietary concentrations >1.0 mg/kg feed and tissue residues >2.0 mg/kg fresh weight should be viewed as presumptive evidence of significant environmental PCP contamination. Data are scarce for PCP and mammalian wildlife; until more data are collected, PCP levels recommended for human health protection (i.e., "no adverse effects" levels) are suggested as reasonable substitutes. In humans, no adverse effects were noted at daily PCP intakes equivalent to 30 ug/kg BW in food, or at concentrations of 21 ug/l in drinking water, 0.5 mg/m^3 in air, 0.5 mg/l in blood plasma, and 1.0 mg/l in blood.

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INTRODUCTION

Pentachlorophenol (PCP) and its water soluble salt, sodium pentachlorophenate, are commercially produced organochlorine compounds used primarily as preservatives of wood and wood products, and secondarily as herbicides, insecticides, fungicides, molluscicides, and bactericides (EPA 1980; Prescott et al. 1982). Both compounds have been sold for these purposes since 1936 under a variety of trade names (Knudson et al. 1974). Because of its widespread use, animals and humans are exposed to significant amounts of PCP; detectable PCP levels are found in most people living in industrialized societies, probably as a result of food chain exposure to PCP-treated wood products (Dougherty 1978; McConnell et al. 1980; Prescott et al. 1982). In Japan, PCP has been widely used as a herbicide in rice fields, but owing to its high toxicity to fishes, its use was limited (beginning in 1971) to upland fields (Nishimura 1984; Mikesell and Boyd 1986). The use of PCP in Japan has resulted in the contamination of all surface water in that country to concentrations of 0.01 to 0.1 ug/l (Lu et al. 1978). The chemical and its degradation products bioconcentrate in fish and are among the phenolic compounds known to taint fish flesh (Boyle et al. 1980). It has been detected in marine fishes and invertebrates, in drinking water, and in human blood and urine (Schimmel et al. 1978; Klemmer et al. 1980; Trujillo et al. 1982). All samples of human milk from nursing mothers tested in Bavaria from 1979 to 1981 contained PCP (Gebefugi and Korte 1983).

In man, illnesses and deaths have been reported after exposure to PCP through diet or by direct contact with PCP-treated products (Prescott et al. 1982). For example, 20 of 80 infants who wore, for 8 days, diapers rinsed in an antimicrobial laundry neutralizer containing sodium pentachlorophenate developed enlarged livers and spleens, had high fevers, and sweated profusely; although most recovered spontaneously, 7 died (Knudsen et al. 1974; EPA 1980). At least 24 industrial PCP fatalities have been reported. The first deaths occurred at a wood preservative plant in France in 1952. Others were recorded at a chemical factory in Japan in 1953, during herbicide spraying in Australia in 1956, at a sawmill in Indonesia in 1958, in South Africa in 1961, and in Canada and the United States in 1965 (Wood et al. 1983). The acute toxic action of PCP in man and experimental animals is caused by the uncoupling of oxidative phosphorylation mechanisms, resulting in marked increases in metabolism (Murphy 1986).

Data are scarce on PCP effects on wildlife, although it is speculated that no wildlife losses should occur under normal PCP application conditions and that chronic toxicity would not be serious because PCP is rapidly excreted

(Bevenue and Beckman 1967). However, mortality was heavy in two species of bats that came into contact with PCP-treated timbers up to 14 months after treatment (Leeuwangh and Voute 1985; Racey and Swift 1986). Furthermore, evidence accumulating on the harmful effects of PCP to domestic animals suggests that the chemical may have considerable adverse effects on other species of wildlife. In the poultry industry, for example, PCP has been implicated in the cause of musty taint in chicken meat and eggs and in increased morbidity in chickens housed on PCP-contaminated wood shavings or given PCP-contaminated food (Prescott et al. 1982). Pentachlorophenol is repellent to animals; diets containing PCP have been rejected by rats, cats, and cattle (Bevenue and Beckman 1967). In farm animals, PCP intoxication has been increasing as a result of confinement in buildings recently treated with a PCP wood preservative, and through dermal contact with PCP-treated fences and feed bunks (Osweiler et al. 1984). Dairy cattle contaminated by PCP produced less milk, grew poorly, and developed skin lesions (Firestone et al. 1979; Greichus et al. 1979; Parker et al. 1980). The issue is confounded by the presence of various amounts of toxic impurities--primarily dioxins and dibenzofurans--in technical and commercial preparations of PCP; these contaminants are mainly responsible for its observed toxicity in rabbits, rats, pigs, cattle, and chickens (Dougherty 1978; McConnell et al. 1980; Prescott et al. 1982). In one example in 1957, millions of chickens died in the southeastern United States after eating poultry feeds containing fat from hides preserved with PCP. Nine dioxins were detected in the toxic animal fat, including the potent 1,2,3,7,8,9-hexachlorodibenzo-p-dioxin isomer (Parker et al. 1980; Stedman et al. 1980; Prescott et al. 1982).

Useful reviews on the ecological and toxicological aspects of PCP have been published by Bevenue and Beckman (1967), Cote (1972), Rao (1978), EPA (1980), Williams (1982), and Choudhury et al. (1986). I here synthesize the available data on environmental aspects of PCP, with emphasis on fish and wildlife. It is part of a continuing series of reviews on chemical contaminants prepared in response to requests for information from environmental specialists of the U.S. Fish and Wildlife Service.

ENVIRONMENTAL CHEMISTRY

GENERAL

Pentachlorophenol and its water soluble salt, sodium pentachlorophenate, are used extensively in agriculture and industry. Most--about 80%--of the 50 million kg of PCP manufactured each year is used in the protection and preservation of wood products. Commercial samples of technical grade PCP are heavily contaminated with many compounds, including chlorophenols, dioxins, dibenzofurans, hexachlorobenzene, and phenoxyphenols; the relative toxicities and accumulation potentials of some of these contaminants may exceed those of PCP by several orders of magnitude. Pentachlorophenol interferes with the production of high energy phosphate compounds essential for cell respiration. In general, it readily degrades in the environment by photochemical, chemical, and microbiological processes.

SOURCES AND USES

Although PCP was first synthesized in 1841, it was not produced commercially until 1936 (Wood et al. 1983; Menzer and Nelson 1986). It has since been registered for use as an insecticide, fungicide, herbicide, algicide, and disinfectant, and as an ingredient in antifouling paint; at least 578 products contain PCP (Cote 1972; Cirelli 1978; Choudhury et al. 1986; Murphy 1986). By 1967, PCP and its sodium salt, sodium pentachlorophenate (Na-PCP), were used extensively in industry and agriculture, due in large part to the solubility of PCP in organic solvents and of Na-PCP in water (Bevenue and Beckman 1967; Cirelli 1978). The major commercial application of technical grade preparations of PCP is in wood preservation formulations, where its fungicidal and bactericidal actions inhibit the growth of wood-destroying organisms (Kinzell et al. 1981).

In the United States, about 80% of the 23 million kg of technical PCP produced annually--or about 46% of worldwide production--is used for wood preservation (Pignatello et al. 1983; Kinzell et al. 1985; Zischke et al. 1985; Choudhury et al. 1986; Mikesell and Boyd 1986). It is the third most heavily used pesticide, preceded only by the herbicides atrazine and alachlor (Kinzell et al. 1981). There are about 470 wood preservative facilities in the United States, scattered among 45 states; they are concentrated in the south, southeast, and northwest--presumably due to the availability of preferred timber species in those regions (Cirelli 1978). Livestock

facilities are often constructed of wood treated with technical PCP; about 50% of all dairy farms in Michigan used PCP-treated wood in the construction of various components of livestock facilities (Kinzell et al. 1985). The chemical is usually applied to wood products after dilution to 5% with solvents such as mineral spirits, No. 2 fuel oil, or kerosene. More than 98% of all wood processed is treated with preservative under pressure; about 0.23 kg of PCP is needed to preserve one cubic foot of wood (Cirelli 1978). Lumber treated with PCP retains its natural appearance, has little or no odor, and can be painted as readily as natural wood (Wood et al. 1983).

In addition to its extensive use by the construction and lumber industries to control damage by mold, termites, powder post beetles, and wood boring insects (Bevenue and Beckman 1967), PCP has been used as a bactericide and fungicide to protect many products, such as adhesives, paper and paperboard, cable coverings, leather, paints, textiles, rope, ink, rubber, and petroleum drilling muds (Bevenue and Beckman 1967; Firestone et al. 1979; Williams 1982). It has been used to control algae and fungi in cooling towers at electric plants (Williams 1982). It has also been added to fabrics for moth proofing, though derivatives such as pentachlorphenol laurate are more widely used for this purpose because their resistance to dry cleaning and washing exceeds that of PCP, and their toxicity to mammals is lower (Bevenue and Beckman 1967). It has been applied in agriculture and around industrial sites as an herbicide and preharvest desiccant, on pastureland, and in pineapple, rice, and sugarcane fields (Bevenue and Beckaman 1967). A Japanese manufacturer has added PCP to soy sauce--in violation of the law--as a preservative (Bevenue and Beckman 1967). It has also been used as a bird repellant: PCP discourages woodpeckers when it is mixed as a pellet and plugged into holes drilled by the bird (Cirelli 1978).

In Canada, the main use of PCP is in the protection and preservation of wood, and secondarily as an herbicide and insecticide for agricultural purposes. A total of 50 wood preserving plants--mostly in British Columbia, Alberta, and Ontario--used about 2.7 million kg of PCP in 1978 (Hoos 1978). Treatment with PCP significantly increased the life of timbers, construction lumber, telephone poles, and railway ties; for example, jackpine poles treated with PCP lasted at least 35 years, compared to 7 years for untreated poles (Hoos 1978).

Sodium pentachlorophenate has been used to control schistosomiasis by eliminating snails that are intermediate hosts of human schistosomes (Bevenue and Beckman 1967). It is also used as a fungicide, bactericide, and algicide in construction materials, emulsion polymers, paints, textiles, and finished paper products; as a preservative for ammonium alginate; and at concentrations of 15 to 40 mg/l, to control microbial growth in secondary oil recovery (Bevenue and Beckman 1967; Cirelli 1978).

PROPERTIES

Pentachlorophenol is readily soluble in most organic solvents, oils, and highly aromatic and olefinic petroleum hydrocarbons--a property that makes it compatible for inclusion in many pesticide formulations (Table 1; Bevenue and Beckman 1967). Purified PCP, however, is practically insoluble in water; therefore, the readily water-soluble sodium pentachlorophenate salt is substituted in many industrial applications (Table 1; Bevenue and Beckman 1967).

The solubility of sodium and potassium pentachlorophenate in water is pH-dependent; it increases from 79 mg/l at pH 5.0 to >4 g/l at pH 8.0 (Bevenue and Beckman 1967). But differential toxicity of PCP in solution is primarily attributable to variations in uptake as a function of pH (Jayaweera et al. 1982; Kaiser and Valdmanis 1982; Fisher and Wadleigh 1986; Smith et al. 1987), and not to water solubility. At pH 4.0, for example, PCP is fully protonated and therefore highly lipophilic, and has its greatest accumulation potential. Conversely, PCP is completely ionized at pH 9.0; lipophilicity is markedly reduced as is its toxicity to the alga Selenastrum capricornutum (Jayaweera et al. 1982; Smith et al. 1987) and the midge, Chironomus riparius (Fisher and Wadleigh 1986).

In recent years it has become clear that many commercial samples of technical grade PCP are heavily contaminated with a large number of potentially toxic compounds and materials (Fig. 1, Table 2). These contaminants include, in part, various isomers of chlorophenols, dibenzofurans, dioxins, hexachlorobenzene, and phenoxyphenols (Table 2), as well as various chlorinated diphenyl ethers, dihydroxybiphenyls, anisoles, catechols, guaiacols, and other chlorinated dibenzodioxin and dibenzofuran isomers (Kaufman 1978; Nilsson et al. 1978; Firestone et al. 1979; EPA 1980; Singh et al. 1985; Menzer and Nelson 1986; Mikesell and Boyd 1986; Murphy 1986; Holsapple et al. 1987). In relative toxicity and accumulation potential, some contaminants in technical grade PCP may exceed the parent compound by several orders of magnitude (Huckins and Petty 1981). For example, some isomers of hexachlorodibenzodioxin, which are present in technical grade PCP at concentrations of 1,000 to 17,300 ug/kg (Table 2), produce LD-50 values in guinea pigs of 60 to 100 ug/kg body weight--thus ranking them as extremely toxic chemicals (Eisler 1986; Murphy 1986).

FATE

Pentachlorophenol may be absorbed into the body through inhalation, diet, or skin contact (Bevenue and Beckman 1967; Williams 1982; Gray et al. 1985). Its acute toxicity results from its ability to interfere with the production of high energy phosphate compounds essential for cell respiration. This interference, or uncoupling, causes stimulation of the cell's metabolism to

Table 1. Chemical and other properties of pentachlorophenol (from Bevenue and Beckman 1967; Cote 1972; Cirelli 1978; EPA 1980; Williams 1982; Hudson et al. 1984; Choudhury et al. 1986; Hill and Camardese 1986; Mayer 1987).

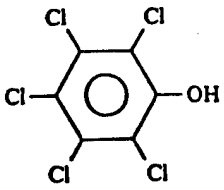
Variable	Datum
Chemical name	Pentachlorophenol, CAS-87-86-5
Alternate names	Chem-Penta, Chemtrol, Chlorophen, Dow Pentachlorophenol, Dowicide 7, Dowicide EC-7, Dowicide G, DP-2, Durotox, Lauxtol A, Ontrack WE-1, PCP, Penchlorol, Penta, Penta General Weed Killer, Pentacon, Penta-kil, Pentanol, Pentasol, Penwar, Permacide, Permaguard, Permasem, Permatox, Priltox, Santobrite, Santophen, Sinituho, Term-l-trol, Weed-Beads, Weedone
Primary uses	Wood preservative, preharvest defoliant, herbicide, molluscicide, insecticide, fungicide
Producers:	Dow Chemical Company, Monsanto Company, Reichold Chemical Company, Vulcan Materials Company
Empirical formula	C_6Cl_5OH
Structural formula	
Physical state	White solid with needle-like crystals. Produced by chlorination of molten phenol. Technical grade material is dark gray to brown.
Molecular weight	266.35
Melting point	190 to 191 ^{o}C
Boiling point	309 to 310 ^{o}C (decomposes)

Table 1 (Concluded)

Variable	Datum
Specific gravity	1.978
Vapor pressure	
25 °C	0.0016 mm Hg
100 °C	0.02 mm Hg
211 °C	40.0 mm Hg
Solubility	
Water	
0 °C	5 mg/l
20 °C	14 mg/l
30 °C	20 mg/l
50 °C	35 mg/l
Carbon tetrachloride	20 to 30 g/l
Benzene	110 to 140 g/l
Ethanol	470 to 520 g/l
Methanol	570 to 650 g/l
Solubility of sodium salt (sodium pentachlorophenate, CAS 131-52-2) in water at 25 °C	330 g/l
Log octanol/water partition coefficient	5.01 to 5.12

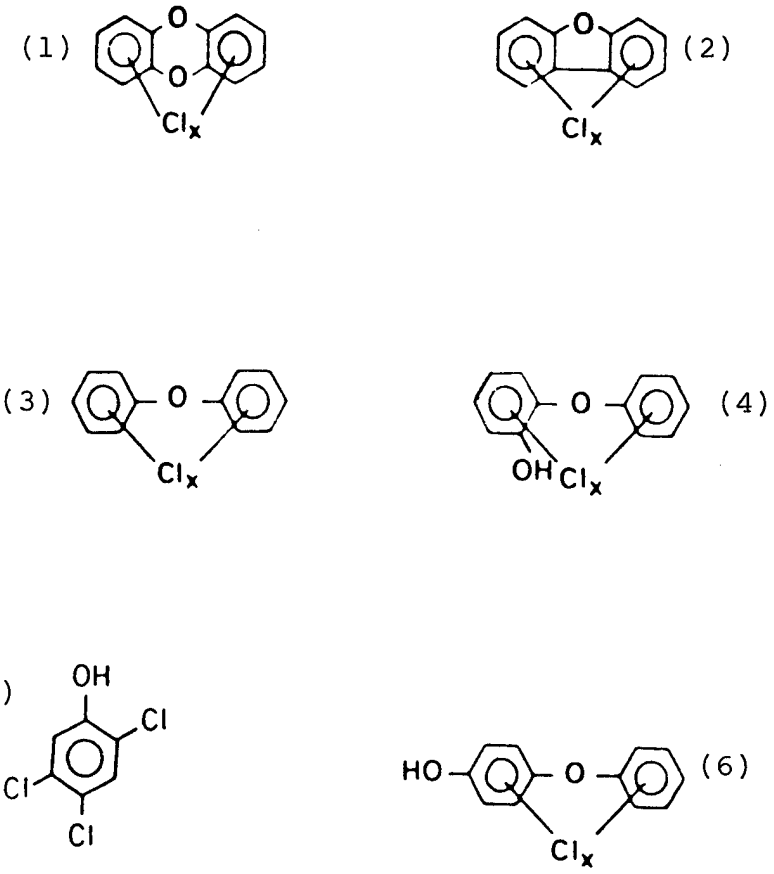


Figure 1. Some impurities found in technical grade pentachlorophenol (from Nilsson et al. 1978). Compounds are: (1) dibenzo-p-dioxins, (2) dibenzofurans, (3) diphenyl ethers, (4) 2- phenoxyphenols, (5) 2, 4,5- trichlorophenol, (6) 4- phenoxyphenols, (7) 2,4- dichlorophenol, (8) 2,4,6- trichlorophenol, (9) 2,3,4,6- tetrachlorophenol.

Table 2. Partial list of contaminants detected in technical grade and purified pentachlorophenol. All values are in mg contaminant/kg product (ppm).

Contaminant	Grade and concentration (% PCP)		Reference[a]
	Technical 85% to 90%	Purified >99%	
CHLOROPHENOLS			
Trichlorophenols[b]	1,000	--	1
Tetrachlorophenols			
2,3,4,6-tetrachlorophenol	49,000	0.25	2,3
2,3,4,5-tetrachlorophenol	9,000	0.073	2,3
Total	40,000 to 80,000	500	1,4
Other chlorophenols[b]	20,000 to 60,000	--	1
DIBENZOFURANS			
Pentachlorodibenzofurans[b]	40	--	5
Hexachlorodibenzofurans[b]	90	--	5
Heptachlorodibenzofurans[b]	400	--	5
Octachlorodibenzofurans[b]	29 to 260	--	5,6
DIOXINS			
Total	1,900 to 2,625	<7	7
Tetrachlorodioxins[b]	0.035 to 0.12	--	1,5
Pentachlorodioxins[b]	0.03	--	5
Hexachlorodioxins[b]	1 to 173	0.00001	1,2,3,4,5,8
Heptachlorodioxins[b]	119 to 1,000	1.8	1,5,6,8
Octachlorodioxins[b]	40 to 4,700	0.0002 to 3.0	1,2,3,4,5,6,8

Table 2 (Concluded)

Contaminant	Grade and concentration (% PCP)		Reference[a]
	Technical 85% to 90%	Purified >99%	
HEXACHLOROBENZENE	56 to 270	0.0014	2,3,6
PHENOXYPHENOLS			
Heptachlorophenoxyphenols[b]	1,200	4.8	6
Octachlorophenoxyphenols[b]	28,000	300	6
Nonachlorophenoxyphenols[b]	15,000	500	6

[a]References: 1, Shull et al. 1986; 2, Zischke et al. 1985; 3, Pignatello et al. 1983; 4, Lamparski et al. 1980; 5, Williams 1982; 6, Cleveland et al. 1982; 7, Eisler 1986; 8, Singh et al. 1985.

[b]Total, when not indicated otherwise.

the toxic stage, which is accompanied by fever and other signs of stress (Bevenue and Beckman 1967; Hodson and Blunt 1981; Williams 1982). The metabolic consequences resemble those of vigorous exercise in some species (Hodson and Blunt 1981). In addition to the proven uncoupling effects on oxidative phosphorylation, the overall inhibitory effects on a variety of enzymes, metabolism of lipids and carbohydrates, ion transport, and protein synthesis may account for the broad spectrum biocidal effects of PCP and its salts (Rao et al. 1979; Gray et al. 1985; Smith et al. 1987). Pentachlorophenol is fetotoxic and teratogenic during early gestation; however, evidence of its mutagenic or carcinogenic properties is incomplete (Williams 1982).

Pentachlorophenol readily degrades in the environment by chemical, microbiological, and photochemical processes (Kaufman 1978; Choudhury et al. 1986). Its suggested metabolic fates include oxidation and dechlorination to tri- and tetrachloro-p-hydroquinones, and glucuronide conjugation to PCP- and tetrachloro-p-hydroquinone conjugates (Williams 1982). In soils, reductive dehalogenation appears to be the most significant PCP degradation pathway, producing mono-, di-, tri-, and tetrachlorophenols, as well as various tetrachlorocatechols and tetrachlorohydroquinones. Further degradation results in ring cleavage, liberation of chloride, and carbon dioxide evolution; degradation is more rapid in flooded or anaerobic soils than in aerobic moist soils (Kaufman 1978). Irradiation of PCP solutions with sunlight or ultraviolet light produces photodegradation products that include chlorinated phenols, tetrachlorodihyroxyl benzenes, and nonaromatic fragments such as dichloromaleic acid (Wong and Crosby 1978; EPA 1980). Subsequent irradiation of the tetrachlorodiols produces hydroxylated trichloro-benzoquinones, trichlorodiols, dichloromaleic acid, and nonaromatic fragments (Wong and Crosby 1978; Boyle et al. 1980). Prolonged irradiation of PCP or its degradation products yielded colorless solutions containing no ether-extractable volatile materials; evaporation of the aqueous layer left no observable polymeric residue (Wong and Crosby 1978). Photolytic condensation of PCP to form octachlorodioxins was observed on a wood substrate. Octachlorodioxin residues ranged from 4 mg/kg for purified PCP, to about 1,500 mg/kg for technical grade PCP (Lamparski et al. 1980).

Pentachlorophenol can be degraded by microbial flora both aerobically and anaerobically; degradation is more rapid under aerobic conditions but slows significantly at temperatures <19 ^{o}C (Pignatello et al. 1985, 1986). Several strains of aerobic bacteria can metabolize and degrade PCP: Flavobacterium sp., a pseudomonad, a coryneform bacterium, and a strain of Arthrobacter (Pignatello et al. 1983; Mikesell and Boyd 1986; Steiert and Crawford 1986; Steiert et al. 1987). Microbial degradation under aerobic or anaerobic conditions was the major process by which PCP was degraded in estuarine sediments; tidal transport and photodegradation played minor roles (DeLaune et al. 1983). The biotic process requires a moderately long adaptive response by the aquatic microflora, but eventually becomes the predominant mechanism of PCP removal (Pignatello et al. 1983, 1985). Several significant observations

were recorded when the degradation and transformation of PCP were documented in freshwater streams continuously dosed with PCP for 16 weeks (Pignatello et al. 1983): photolysis accounted for a 5% to 28% decline in initial PCP concentrations and was most rapid at the water surface under conditions of bright sunlight; adsorption to sediments and uptake by biota accounted for less than 5% loss in acclimated waters and probably less than 15% in unacclimatized waters; and microbial degradation of PCP became significant about 3 weeks after dosing and eventually became the primary mechanism of PCP removal, accounting for up to a 46% decline in initial PCP.

The half-life ($Tb\frac{1}{2}$) of PCP in water ranged from 0.15 to 15 days; degradation was most rapid under conditions of high incident radiation, high dissolved oxygen, and elevated pH (Bevenue and Beckman 1967; Wong and Crosby 1978; Boyle et al. 1980; Niimi and Cho 1983; Crossland and Wolff 1985; Smith et al. 1987). The $Tb\frac{1}{2}$ in the water column controlled by microbial degradation alone is usually 5 to 12 hours (Pignatello et al. 1986). Technical grade PCP was initially degraded at the same rate as reagent grade PCP by anaerobic microorganisms in municipal sewage sludge, but was later degraded more slowly. Dechlorination and mineralization (to carbon dioxide and methane) of the reagent grade PCP was complete in 7 to 9 days, but only half the technical grade PCP had been transformed in 6 to 10 days (Mikesell and Boyd 1986).

In soils, PCP persisted for 15 to more than 60 days, depending on soil conditions and application rate. At initial concentrations of 100 mg PCP/kg soil, the $Tb\frac{1}{2}$ was 10 to 40 days at 30 ^{o}C under flooded conditions; however, in aerobic soils there was virtually no degradation after 2 months (Kaufman 1978). In rice paddy soils, initial concentrations of 4 mg PCP/kg fell to 2 mg/kg in 7 days (Bevenue and Beckman 1967). Pentachlorophenol was still measurable after 12 months in warm, moist soils (Cote 1972; EPA 1980). In estuarine sediments, degradation was most rapid under conditions of increased oxygen and a pH of 8.0 (DeLaune et al. 1983).

Pentachlorophenol solutions in water at the appropriate pH and dissolved oxygen content decompose in sunlight, and this makes a strong case for the likelihood of essentially total PCP destruction in aquatic environments (Wong and Crosby 1978). The short residence time of PCP in an aquatic system before degradation further suggests that biological effects would be most pronounced in localized areas that receive PCP continuously from a point source (Nimmi and Cho 1983).

BACKGROUND CONCENTRATIONS

GENERAL

Measurable PCP concentrations in field collections of living and nonliving materials over widespread geographic areas are almost certainly due to anthropogenic activities, especially to the use of the chemical as a wood preservative.

BIOLOGICAL AND NONBIOLOGICAL SAMPLES

Pentachlorophenol-contaminated air, rain, snow, surface waters, drinking waters, groundwaters, and aquatic biota are common in the United States (Table 3; Pignatello et al. 1983; Choudhury et al. 1986). Residues of PCP in food, water, and mammalian tissues may result from the direct use of PCP as a wood preservative and pesticide or as a result of use of other chemicals that form PCP as degradation products--i.e., hexachlorobenzene and lindane (EPA 1980; Choudhury et al. 1986). To confound matters, PCP was judged to be the source of dioxin and dibenzofuran contamination in chickens in Canada (Ryan et al. 1985). More than 50% of all chickens sampled contained hexachlorinated dibenzo-p-dioxins (hexa CDDs) at concentrations of 27 ng/kg fat and higher; 62% contained hepta CDDs at more than 52 ng/kg, and 46% contained octa CDDs at more than 90 ng/kg; concentrations of hexa-, and heptachlorinated dibenzofurans were similar.

Pentachlorophenol was found at high concentrations in all samples of sediments, waters, and biota collected near industrial facilities that used PCP as a wood preservative (Niimi and Cho 1983; Table 3). Fish can bioconcentrate PCP from water up to 10,000 times (Fox and Joshi 1984). However, similar concentrations were measured in blue mussel, Mytilus edulis (Folke and Birklund 1986), and softshell clam, Mya arenaria (Butte et al. 1985), from the vicinity of PCP-contaminated wastewater discharges as well as from more distant collection sites; thus PCP bioaccumulation in marine bivalve molluscs does not appear to be dose related.

Table 3. Pentachlorophenol concentrations in nonbiological and living materials.

Compartment and units	Concentration	Reference[a]
NONBIOLOGICAL		
Aquatic (ug/l)		
Rivers, Southwest Japan	1 to 10	1
Willamette River, Oregon	0.1 to 0.7	1
Waters of British Columbia	up to 7.3	1
Drinking water	0.06	2,3
Northern California		
Moss Landing, seawater	<1	4
Sacramento, sewage discharge	<1	4
Oroville		
Drainage water	20	4
Drinking water	227 (1 to 800)	4
Surface water	0.7	3
Near wood preserving facility, Bay of Quinte, Lake Ontario, 1978		
Surface film	5.8	5
Water column	5.7	5
Air (ng/m^3)		
Uninhabited mountainous area	~0.25	6
Rooms containing PCP-treated wood or paint	up to 160	6
Near PCP wood preservative facility	Usually 263 to 1,188; Max. 297,000	2
PCP pressure treating room	Max. 15,000	2
PCP storage areas	9 to 9,000	2
Sediments (ug/kg dry weight)		
Bay of Quinte, Lake Ontario, 1978	60	5

Table 3. (Continued)

Compartment and units	Concentration	Reference[a]
BIOLOGICAL		
Freshwater organisms (ug/kg fresh weight)		
Lake Ontario, western basin		
Fish, whole less intestine		
Rainbow trout, *Salmo gairdneri*	24 (10 to 39)	7
Lake trout, *Salvelinus namaycush*	Max. 11	7
Coho salmon, *Oncorhynchus kisutch*	Max. 21	7
Brown trout, *Salmo trutta*	6, Max. 11	7
Rainbow smelt, *Osmerus mordax*	Max. 0.5	7
Alewife, *Alosa pseudoharengus*	Max. 0.3	7
Bay of Quinte, Lake Ontario, 1978		
Fish, whole		
Brown bullhead, *Ictalurus nebulosus*	260	5
Yellow perch, *Perca flavescens*	155	5
Invertebrates		
Annelids	Max. 85	5
Chironomids	Max. 1	5
Alga		
Cladophora sp.	7	5
Marine organisms (ug/kg)		
Blue mussel, *Mytilus edulis*		
Denmark, 1985, soft parts		
Fresh weight	5 to 33	8
Dry weight	32 to 244	8
Lipid weight	398 to 3473	8

Table 3. (Continued)

Compartment and units	Concentration	Reference[a]
Wildlife (mg/kg whole mummified body)		
Dutch pond bat, *Myotis dasycneme*		
Netherlands, found dead		
Berlikum roost (treated with PCP)		
1974	8 to 36	9
1977	410 to 795	9
1978	746 to 1,105	9
1979	10 to 283	9
Tjerkwerd roost (Control site)		
1978	<7	9
1979	<4	9
Livestock, Canada (ug/kg)		
Fat, chicken and pork		
Lipid weight	Usually (60% frequency) >10	10
Liver, pig		
Fresh weight	Always >50	10
Man (ug/kg fresh weight)		
Unexposed		
Urine	2 to 11	2,11
Blood serum	4 to 10	2,11
Adipose tissue	12 to 52	11
Milk, nursing mothers, Bavaria, 1979-1981	0.67 (0.03 to 2.8)	10
Exposed		
Urine	80 to 300	2
Blood serum	1,000 to 2,000; Max. 3,900	2
Daily diet (ug)	1 to 6	12

Table 3. (Concluded)

[a] References: 1, Dominguez and Chapman 1984; 2, EPA 1980; 3, Menzer and Nelson 1986; 4, Wong and Crosby 1978; 5, Fox and Joshi 1984; 6, Pignatello et al. 1983; 7, Niimi and Cho 1983; 8, Folke and Birklund 1986; 9, Leeuwangh and Voute 1985; 10, Ryan et al. 1985; 11, Gebefugi and Korte 1983; 12, Choudhury et al. 1986.

LETHAL AND SUBLETHAL EFFECTS

GENERAL

The toxicity of commercial or technical grades of PCP significantly exceeds that of analytical or purified PCP. Some of this added toxicity is attributed to impurities such as dioxins, dibenzofurans, chlorophenols, and hexachlorobenzene. Pentachlorophenol is rapidly accumulated and rapidly excreted, and has little tendency to persist in living organisms. It acts by uncoupling oxidative phosphorylation.

Terrestrial plants and soil invertebrates were adversely affected at 0.3 mg PCP/l (root growth), and at 1 to 5 g PCP/m^2 soil (reduction in soil biota populations).

Pentachlorophenol was most toxic and most rapidly metabolized in aquatic environments at elevated temperatures and reduced pH. Adverse effects on growth, survival, and reproduction of representative sensitive species of aquatic organisms occurred at PCP concentrations of about 8 to 80 ug/l for algae and macrophytes, about 3 to 100 ug/l for invertebrates (especially molluscs), and <1 to 68 ug/l for fishes, especially salmonids.

Fatal PCP doses for birds were 380 to 504 mg/kg BW (acute oral), >3,850 mg/kg in diets, and >285 mg/kg in nesting materials. Adverse sublethal effects were noted at dietary levels as low as 1.0 mg/kg ration. Residues (mg/kg fresh weight) in birds found dead from PCP poisoning were >11 in brain, >20 in kidney, >46 in liver, and 50 to 100 in egg.

Data are scarce on the toxicity of PCP to mammalian wildlife, but studies with livestock and small laboratory animals show that the chemical is rapidily excreted. However, there is great variability between species in their ability to depurate PCP, as well as in their overall sensitivity. Acute oral LD-50's in laboratory animals were 27 to 300 mg/kg BW. Tissue residues were elevated at dietary levels as low as 0.05 mg/kg feed and at air levels >0.1 mg/m^3. Histopathology, reproductive impairment, and retarded growth were evident at doses of 0.2 to 1.25 mg/kg BW, and when the diets fed contained >30 mg PCP/kg.

TERRESTRIAL PLANTS AND INVERTEBRATES

Pentachlorophenol is toxic to plant mitochondria; the mode of action is similar to that in other organisms--i.e., uncoupling of oxidative phosphorylation. At 267 ug PCP/l, 50% uncoupling was noted in isolated mitochondria of potato, Solanum tuberosum, and mung bean, Phaseolus aureus (Ravanel and Tissut 1986). Both PCP and its metabolite tetrachlorohydroquinone adversely affect cell growth and synthesis of RNA and ribosome in yeast, Saccharomyces sp., in a dose-related manner (Ehrlich et al. 1987).

Uptake of PCP by rice (Oryza sativa) grown over a 2-year period under flooded conditions was studied after a single application of radiolabeled PCP was applied to the soil at 23 kg/ha (Weiss et al. 1982). During the first year, PCP uptake was 12.9% of the application. Roots contained about 5 mg PCP/kg, distributed as follows (mg/kg): 3.95 as unextractable residues, 0.48 as polar nonhydrolyzable substances, 0.43 as free and conjugated lower chlorinated phenols, 0.14 as free PCP, 0.07 as anisoles, 0.06 as conjugated PCP, 0.03 as hydroxymonomethoxytetrachlorobenzenes, and 0.01 as dimethoxytetrachlorobenzenes. In the second year, PCP uptake was reduced to 2.5%, and soil residues corresponded to 8.4 kg/ha; the amounts of unextractable residues in plants increased, and lower chlorinated conjugated phenols were identified (Weiss et al. 1982). Root growth in rice seedlings was inhibited 50% at 0.3 mg PCP/l (Nagasawa et al. 1981).

Pentachlorophenol applied to beech forest soils every 2 months for 2 years at the rate of 1.0 g/m^2 markedly reduced populations of soil organisms; at 5.0 g/m^2, it drastically reduced most of the soil animal species, and also the microflora (Zietz et al. 1987). Reduction of the soil metabolism by PCP retards decomposition and affects the overall nutrient balance of forest ecosystems (Zietz et al. 1987).

AQUATIC BIOTA

Pentachlorophenol affects energy metabolism by partly uncoupling oxidative phosphorylation and increasing oxygen consumption, by altering the activities of several glycolytic enzymes and the citric acid cycle enzymes, and by increasing the consumption rate of stored lipid (Johansen et al. 1985; Brown et al. 1987; McKim et al. 1987). Collectively, these events could reduce the availability of energy for maintenance and growth and thereby reduce the survival of larval fish and the ability of prey to escape from a predator (Brown et al. 1985, 1987).

The accumulation of PCP in fishes is rapid, and primarily by direct uptake from water rather than through the food chain or diet (Niimi and Cho 1983). Signs of PCP intoxication in fish include rapid swimming at the surface and increased opercular movements, followed by loss of balance, settling to the bottom, and death (Holmberg et al. 1972; Gupta 1983). The PCP is rapidly excreted by fishes after conjugates of PCP-glucuronide and PCP-sulfate are formed; half-lives in tissues are less than 24 hours (EPA 1980). Major roles were played by gall bladder and bile in PCP-glucuronide depuration kinetics, and by gill in PCP-sulfate depuration (Kobayashi 1978; Lech et al. 1978; McKim et al. 1986). It has been suggested that the efficient elimination of PCP should allow vertebrates to tolerate periodic low doses of PCP without toxic effects (McKim et al. 1986).

Many species of aquatic organisms were found dead in rice fields of Surinam, South America, after they were sprayed with PCP to control populations of snails (Vermeer et al. 1974). Residues of PCP in dead organisms (mg PCP/kg fresh body weight) were 8.1 in frogs (Pseudis paradoxa); 36.8 in snails (Pomacea spp.); and, in three species of fish, 31.2 in krobia (Cichlasoma bimaculatum), 41.6 in kwi kwi (Hoplosternum littorale), and 59.4 in srieba (Astyanax bimaculatus). Pentachlorophenol was also implicated in fish kills in Europe and North America, all of which were associated with the pulpwood industry (Schimmel et al. 1978). In December 1974, near Hattiesburg, Mississippi, water containing PCP in fuel oil that overflowed the banks of a holding pond of a wood-treatment waste water facility killed all fish in a 24 ha lake. Concentrations of PCP in water and fish returned to background concentrations 10 months after the spill; however, the chemical persisted in leaf litter and sediments for at least 17 months after the spill (Pierce and Victor 1978). In December 1976, another fish kill was observed near the same facility. Residues of PCP in surviving fish--including bluegills (Lepomis macrochirus), largemouth bass (Micropterus salmoides), and channel catfish (Ictalurus punctatus)--were greatly elevated one month later: 8 to 19 mg PCP/kg fresh weight in muscle, 42 to 48 mg/kg in gill, and 130 to 221 mg/kg in liver (Pierce and Victor 1978). Pentachlorophenol persisted in fish for 6 to 10 months before reaching background concentrations.

Studies with experimental ecosystems have indicated that the effects of PCP on community structure and activity are profound. These included a reduction in the number of individuals and species of estuarine macrobenthos after exposure to 55 to 76 ug PCP/l for 5 to 9 weeks (Tagatz et al. 1977, 1983) or 15.8 ug/l for 13 weeks (Tagatz et al. 1978); a decrease in periphyton biomass, fish growth, and larval drift, and a suppression of community metabolism at 48 ug PCP/l after 3 months exposure (Hedtke and Arthur 1985; Zischke et al. 1985; Yount and Richter 1986); elevated levels of PCP in postlarval shrimp, Penaeus vannamei, after chronic exposure to 10 ug PCP/l (Seidler et al. 1986); and bioconcentration factors after exposure to radiolabeled PCP of 5X for an alga (Oedogonium cardiacum), 21X for a snail (Physa sp.), 26X for a mosquito larva (Culex pipiens quinquefasciatus), 132X for mosquitofish (Gambusia affinis), and 205X for Daphnia magna (Lu et al.

1978).

In laboratory studies, increased accumulation and adverse effects on growth, survival, and reproduction were seen in sensitive species of aquatic organisms: in algae and macrophytes at water concentrations (ug PCP/l) of 7.5 to 80; in a wide variety of invertebrates, especially molluscs, at 2.5 to 100; and in fishes, especially salmonids, at <1.0 to 68 (Table 4).

Biocidal properties of PCP were significantly modified by water pH and temperature, and by the purity of PCP compounds tested. In general, PCP was most toxic and was metabolized most rapidly (Table 4) at elevated water temperatures (EPA 1980; Hodson and Blunt 1981; Nimmi and Palazzo 1985; Fisher 1986) and at reduced pH (Bevenue and Beckman 1967; EPA 1980; Dave 1984; Choudhury et al. 1986; Fisher 1986; Seidler et al. 1986). Increasing pH of the water column decreases the hazard of PCP to aquatic biota: at pH above 4.8, for example, hydroxyl proton is dissociated and penetration in aquatic organisms is reduced (Fisher 1986). All authorities agree that commercial or technical grades of PCP are significantly more toxic to aquatic organisms than is purified PCP (EPA 1980; Cleveland et al. 1982; Huckins and Petty 1983; Dominquez and Chapman 1984; Stuart and Robinson 1985; Hamilton et al. 1986; Nagler et al. 1986). The sublethal effects of low concentrations of commercial PCP to aquatic biota are due primarily to impurities composed mostly of octa- and nonachlorophenoxyphenols (Hamilton et al. 1986), and also to relatively large quantities of hexachlorobenzene, dioxins, and dibenzofurans (Cleveland et al. 1982).

BIRDS

Signs of PCP intoxication in birds include excessive drinking and regurgitation, rapid breathing, wing shivers or twitching, jerkiness, shakiness, ataxia, tremors, and spasms (Hudson et al. 1984). Signs sometimes appear within 10 minutes. Mallards usually die 2 to 24 hours posttreatment, and ring-necked pheasants 3 to 5 days posttreatment; remission in pheasants requires up to 2 weeks (Hudson et al. 1984).

Pentachlorophenol killed various species of birds at single oral doses of 380 to 504 mg/kg BW, at dietary concentrations of 3,850 mg/kg ration fed over a 5-day period, and when nesting materials contained >285 mg/kg. Residues (mg/kg fresh weight tissue) in birds found dead from PCP poisoning were 11 in brain, 20 in kidney, 46 in liver, and 50 to 100 in egg (Table 5). Sublethal effects, including liver histopathology and diarrhea, were reported in domestic chickens at dietary levels as low as 1 mg PCP/kg feed over an 8-week period; significant accumulations in tissues were measured after consumption for 14 days of diets containing 10 mg PCP/kg (Table 5). Residues in chickens fed PCP-containing diets for 8 weeks were dose-related and highest in kidney

Table 4. Lethal and sublethal effects of pentachlorophenol on selected species of aquatic organisms.

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
ALGAE AND MACROPHYTES			
Alga, *Chlorella pyrenoidosa*	7.5	Total chlorosis inhibition in 72 hours.	1
Alga, *Skeletonema costatum*	17 to 20	50% reduction in cell numbers at 96 hours.	1
Filamentous algae (*Chara* sp., *Enteromorpha* sp.)	50 to 100	Lethal in 30 days in outdoor ponds; Their decay was responsible for depression of dissolved oxygen and later fish deaths.	2
Alga, *Scenedesmus costatum*	80	50% growth inhibition in 96 hours.	2
Alga, *Selenastrum capricornutum*	110 to 150	50% growth reduction in 96 hours in soft water.	3
S. capricornutum	290	50% growth inhibition in 96 hours.	2
S. capricornutum	760	50% growth reduction in 96 hours in hard water.	3
Alga, *Dunaliella tertiolecta*	170 to 206	50% reduction in cell numbers in 96 hours.	1

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
Alga, Thalassiosira pseudonana	179 to 189	50% reduction in cell numbers in 96 hours.	1
Duckweed, Lemna minor	800	50% inhibition of chlorosis in 48 hours.	1
L. minor	1,000	Inhibition of photosynthetic oxygen production.	5
L. minor	1,400	No measurable effect after exposure for 21 days.	4
INVERTEBRATES			
Eastern oyster, Crassostrea virginica	2.5	BCF of 78X in 28 days; 100% depuration within 4 days postexposure.	6
C. virginica	25	BCF of 41X between days 4 and 28 of exposure; 100% depuration within 4 days postexposure.	6
C. virginica	34	50% reduction in shell deposition in 8 days.	1
C. virginica	40	50% abnormal development of larvae in 48 hours.	7
C. virginica	77	LC-50 (96 hours).	1

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
Cladoceran, Ceriodaphnia reticulata	4.1	Reduction in number of young produced per female in lifetime exposure.	4
C. reticulata	164	LC-50 (48 hours).	8
Polychaete worm, Ophryotrocha diadema	10 to 13	No effect on reproduction in 41 days.	8
O. diadema	23 to 75	50% inhibition in reproduction in 41 days.	8
Clam, Mulinia lateralis	15.8	Populations reduced after 7 days.	9
Razor clam, Ensis minor	15.8	Populations reduced after 7 days.	9
Hydra, Hydra littoralis	19	Threshold for reproduction inhibition.	8
Snail, Physa gyrina	26	Reduction in number of egg masses produced and in egg survival during lifetime exposure.	4,10
P. gyrina	220	LC-50 (96 hours) at 24 ^{o}C.	4
P. gyrina	730	LC-50 (96 hours) at 8.6 ^{o}C.	11
P. gyrina	1,380	LC-50 (96 hours) at 3.2 ^{o}C.	4
Pacific oyster, Crassostrea gigas	55	62% of exposed embryos developed abnormally in 48 hours.	1

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
Midge, Chironomus riparius, 4th instar	84	LC-50 (24 hours) at pH 4.	12
C. riparius	465	LC-50 (24 hours) at pH 6.	12
C. riparius	631	50% locomotion inhibition at 35 °C.	13
C. riparius	1,176	50% locomotion inhibition at 15 °C.	13
C. riparius	1,948	LC-50 (24 hours) at pH 9.	12
Snail, Lymnaea acuminata	100	LC-16 (96 hours).	14
L. acuminata	160	LC-50 (96 hours).	14
L. acuminata	210	LC-84 (96 hours).	14
Snail, Australorbis glabratus	100	Reduction in egg production and viability after exposure for 7 to 8 days.	2
Sea urchin, Paracentrotus lividus, embryos	100	Reductions in various amino acid activity levels during exposure for 40 hours.	15
P. lividus	200	Number and size of swimming blastulae, gastrulae, and plutei reduced in 40-hour exposure.	15
Short-necked clam, Tapes philippinarum	100	Lethal in 120 hours.	16

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
Snail, Lymnaea luteola	112	LC-50 (96 hours).	17
Flatworm, Pugesia lugubris	130	LC-50 (48 hours).	8
Snail, Lymnaea stagnalis	180	LC-50 (16 days).	2
Cladoceran, Daphnia magna	180	No observable effect level in lifetime exposure.	1
D. magna	320	Some adverse effects observed in lifetime exposure.	1
D. magna	370 to 440	50% immobilization in 48 hours.	18
D. magna	475	LC-50 (96 hours).	1
Cladoceran, Simocephalus vetulus	204	LC-50 (96 hours) at 24 ^{o}C.	4
S. vetulus	670	LC-50 (96 hours) at 18 ^{o}C.	11
Tubificid worm, Tubifex tubifex	286	LC-50 (24 hours) at pH 7.5.	1
T. tubifex	619	LC-50 (24 hours) at pH 8.5.	1
T. tubifex	1,294	LC-50 (24 hours) at pH 9.5.	1

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
Snail, Gillia altilis	300	LC-50 (96 hours) in flow-through test.	19
G. altilis	810	LC-50 (96 hours) in static test.	19
Mysid, Mysidopsis bahia	320	LC-50 (96 hours).	20
Grass shrimp, Palaemonetes pugio	400 to 440	LC-50 (96 hours) during molting.	20,21
P. pugio	473 to 637	Adversely affects initiation and progress of limb regeneration without altering duration of molt cycle.	22
P. pugio	649 to 1,200	LC-50 (96 hours).	1,7
P. pugio	1,000	Histopathology of gill, hepatopancreas, and midgut epithelial cells after exposure for 12 days in shrimp that molted; normal tissues in shrimp that had not yet molted.	21
P. pugio	2,500	LC-50 (96 hours) during intermolt.	20,21
Polychaete worm, Neanthes arenaceodentata	435	LC-50 (96 hours).	1
Amphipod, Gammarus pseudolimnaeus	770	Significant decreases in free amino acids after 5 days; in whole body glycogen, protein, and caloric content after 15 days; and in lipid content after 30 days.	23

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
G. pseudolimnaeus	860	LC-50 (30 days).	23
G. pseudolimnaeus	1,150	LC-50 (96 hours).	24
G. pseudolimnaeus	1,680	At 48 hours, significant decrease in total free amino acid levels.	24
Pond snail, Viviparus bengalensis	340	LC-50 (96 hours).	25
Caddisfly, Philarctus quaeris	1,200	LC-50 (96 hours).	11
Mayfly, Callibaetes skokianus	1,700	LC-50 (96 hours).	11
Amphipod, Crangonyx pseudogracilis	1,900	LC-50 (96 hours).	11
Isopod, Asellus racovitzai	2,300	LC-50 (96 hours) at 8.6 ^{o}C.	11
A. racovitzai	4,320	LC-50 (96 hours) at 4.2 ^{o}C.	4
A. racovitzai	>7,770	LC-50 (96 hours) at 3.2 ^{o}C.	4
Crayfish, Astacus fluviatilis	9,000	LC-50 (8 days) at pH 6.5.	26
A. fluviatilis	53,000	LC-50 (8 days) at pH 7.5.	26

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
VERTEBRATES			
Rainbow trout, Salmo gairdneri	0.035	After 115 days, whole fish BCF 286X to 572X; half eliminated in 7 days.	8,57
S. gairdneri	0.7	After 115 days, BCF of 160X in whole fish; $Tb\frac{1}{2}$ elimination time of 7 days.	8,57
S. gairdneri	1.0	Whole body BCF of 221X in 18 hours and 466X in 11.7 days; $Tb\frac{1}{2}$ of 65 hours, and 95% depuration in 12 days. Highest PCP tissue residues were bile >> liver >> blood > kidney > spleen > skin-bone-gill-gonad > muscle.	27
S. gairdneri	7.4	27% inhibition of growth in 28 days.	1
S. gairdneri	10 to 20	Some deaths during exposure from fertilization to yolk-sac absorption; 100% dead at 5 mg dissolved oxygen(DO)/l and 20 ug PCP/l; 100% dead at 3 mg DO/l and 10 ug PCP/l.	28
S. gairdneri	11	No adverse effects observed during exposure from fertilization through day 72.	29
S. gairdneri	19	Embryo mortality negligible during exposure from fertilization through day 72. Alevin mortality 3X greater than in controls. Growth in length and weight reduced. Fin erosion, mild cranial malformations, and lethargy reported.	29

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
S. gairdneri	22	48% reduction in viable oocytes after exposure for 18 days.	30
S. gairdneri	25	After 24 hours, BCF of 40X in muscle, 240X in fat, 260X in blood, and 640X in liver. $Tb\frac{1}{2}$ values ranged from 7 hours in muscle to 23 hours in fat.	31,58
S. gairdneri	28	11% to 19% inhibition in growth in 20 to 38 days.	1
S. gairdneri	34 to 121	LC-50 (96 hours).	1,29,32,33,34
S. gairdneri	40	100% mortality during exposure from fertilization to yolk-sac absorption (72 days).	28
S. gairdneri	46	LC-100 (41 days).	1
S. gairdneri	49	81% reduction in oocytes available to complete oogenesis after exposure for 18 days.	30
S. gairdneri	60	Eye abnormalities noted in developing embryos after exposure for 17 days; all dead at day 72 of exposure.	29
S. gairdneri	10,000	LC-50 (3.5 hours).	35
Sockeye salmon, Oncorhynchus nerka	3.2	10% growth inhibition in 6 weeks.	1

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
O. nerka	63 to 68	LC-50 (96 hours).	1,56
Common carp, Cyprinus carpio	9.5	LC-50 (96 hours), larvae.	8
C. carpio	266	Reduction in liver glucose and glycogen release after 3 days; high accumulations in liver.	36
C. carpio	1,500	LC-50 (3 hours).	37
Largemouth bass, Micropterus salmoides	10	Food conversion efficiency significantly reduced in a concentration-dependent fashion at concentrations >10 ug/l.	38
M. salmoides	25.2	Growth reduction after exposure for 52 days.	38
M. salmoides	45	Decline in feeding activity after 8 weeks	38
M. salmoides	50	After 14 days, food conversion efficiency reduced 30%, reduction in ability to capture prey and in food consumed.	39
M. salmoides	54	LC-50 (120 days).	38
M. salmoides	67	Exposure for 8 weeks produced hyperactivity and reductions in feeding rate and in prey capture.	40
M. salmoides	136 to 287	LC-50 (96 hours).	38,39,41

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
Rasbora, Rasbora daniconius neilgeriensis	10	LC-0 (96 hours).	42
R.d. neilgeriensis	67	LC-16 (96 hours).	42
R.d. neilgeriensis	148	LC-50 (96 hours).	42
R.d. neilgeriensis	330	LC-84 (96 hours).	42
Pinfish, Lagodon rhomboides	31 to 53	LC-50 (96 hours).	1,7,20
Bluegill, Lepomis macrochirus	32 to 215	LC-50 (96 hours).	1,11,32,33
L. macrochirus	100	After exposure for 8 days, BCF 13X in muscle, 60X in gill, 210X in gastrointestinal tract, and 350X in liver. Rapid elimination, but some residues in muscle and liver were detectable 16 days postapplication.	43
L. macrochirus	100	Liver histopathology after 32 days; degenerative changes detectable after 2 days.	44
L. macrochirus	432	LC-100 (8 days).	10
Fathead minnow, Pimephales promelas	45	No observable effect level in lifetime exposure.	1,54
P. promelas	48	Growth and larval drift reduced after exposure for 12 weeks.	10

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
P. promelas	50	Whole body BCF of 174X after 14 days; nondetectable residues after 14 days in clean water.	45
P. promelas	73	Some adverse effects in lifetime exposure.	1,54
P. promelas	85	Growth reduction after 90 days exposure.	46
P. promelas	120	LC-50 (96 hours) at 16.6 ^{o}C.	4
P. promelas	170	LC-50 (96 hours) at 10.1 ^{o}C.	4
P. promelas	200 to 350	LC-50 (96 hours).	2,11,35
P. promelas	300	LC-50 (96 hours) at 3.4 ^{o}C.	4
Atlantic salmon, Salmo salar	46	Altered temperature preference in 24 hours.	1
S. salar	500	LC-50 (96 hours).	35
S. salar	2,000	LC-50 (10.5 hours).	35
S. salar	10,000	LC-50 (2.7 hours).	35
Sheepshead minnow, Cyprinodon variegatus	47	No observable effect level in lifetime exposure.	1
C. variegatus	88	12% mortality in lifetime exposure.	47

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
C. variegatus	195	Life cycle exposure resulted in reduced hatch and survival of second generation fish.	47
C. variegatus	223 to 392	LC-50 (96 hours) for fry age 1 day to 6 weeks.	7
C. variegatus	389	LC-100 (60 days).	47
C. variegatus	442	LC-50 (96 hours).	1,47
Flounder, Pleuronectes platessa	50	LC-50 (8 weeks), eggs.	8
P. platessa	60 to 140	LC-50 (96 hours), larvae.	8
P. platessa	100 to 130	LC-50 (96 hours), juveniles.	8
Channel catfish, Ictalurus punctatus	54 to 68	LC-50 (96 hours).	8,32,33
Coho salmon, Oncorhynchus kisutch	55	LC-50 (96 hours).	1
White crappie, Pomoxis annularis	56 to 75	LC-50 (96 hours).	35
Longnose killifish, Fundulus similis	57 to 610	Whole body BCF of 53X after 168 hours; $Tb_{\frac{1}{2}}$ of 4.7 days. Whole body residues up to 33 mg/kg fresh weight.	48

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
Chinook salmon, Oncorhynchus tshawytscha	68 to 78	LC-50 (96 hours).	1,32,55
White sucker, Catostomus commersoni	85	LC-50 (96 hours).	4
Eel, Anguilla anguilla	100	Seawater-exposed eels, after exposure for 8 days, had 33 mg PCP/kg fresh weight in liver, 9 in muscle, and 4 in blood; after depuration for 8 days, values were 12 in liver, 4 in muscle, and 2 in blood. Eels exposed in freshwater for 4 days had 2 to 9 mg PCP/kg tissue, and <1.2 mg/kg 38 days postexposure.	49
Goldfish, Carassius auratus	100	BCF of 1,000X after 12 hours. Residues in dead fish were 82 to 116 mg PCP/kg BW; no surviving fish contained more than 114 mg/kg.	16,48
C. auratus	200	Whole body residue of 116 mg/kg after exposure for 120 hours.	48
Tilapia, Tilapia nilotica	100	In 24-hour tests, fish acclimatized to seawater were twice as resistant as freshwater-acclimatized fish to biocidal PCP properties, and contained lower residues.	50
Mullet, Rhinomugil corsula	100	Metabolic rate elevated after 3 hours.	37

Table 4. (Continued)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
R. corsula	1,000	LC-50 (3 hours).	37
Striped mullet, Mugil cephalus	112	LC-50 (96 hours), whole body BCF of 38X.	1,6
Brook trout, Salvelinus fontinalis	128	LC-50 (96 hours).	1
Salamander, Ambystoma mexicanus	130	LC-0 (48 hours).	8
A. mexicanus	300	LC-50 (48 hours).	8
Common shiner, Notropis cornutus	180	25% growth reduction after exposure for 7 days.	51
N. cornutus	320	LC-100 (7 days).	51
Fish, 19 species	200 to 600	LC-50 (96 hours).	35
Frog, Xenopus laevis	210	LC-0 (48 hours).	8
X. laevis	260	LC-50 (48 hours).	8
Guppy, Poecilia reticulata	500 to 700	Reduction in ability to escape from piscine predators.	52
P. reticulata	700	LC-21 (30 days).	52

Table 4. (Concluded)

Species and other variables	Concentration, in ug PCP/l medium	Effect	Reference[a]
P. reticulata	1,000	LC-0 (12 days); possible acclimatization.	53
P. reticulata	1,020	LC-50 (96 hours).	52
Sea lamprey, Petromyzon marinus	924	LC-100 (4 hours).	1

[a]References: 1, EPA 1980; 2, Crossland and Wolff 1985; 3, Smith et al. 1987; 4, Hedtke et al. 1986; 5, Huber et al. 1982; 6, Schimmel et al. 1978; 7, Borthwick and Schimmel 1978; 8, Choudhury et al. 1986; 9, Tagatz et al. 1981; 10, Zischke et al. 1985; 11, Hedtke and Arthur 1985; 12, Fisher and Wadleigh 1986; 13, Fisher 1986; 14, Gupta and Rao 1982; 15, Ozretic and Krajnovic-Ozretic 1985; 16, Kobayashi 1978; 17, Gupta et al. 1984; 18, Berglind and Dave 1984; 19, Stuart and Robinson 1985; 20, Mayer 1987; 21, Rao and Doughtie 1984; 22, Rao et al. 1978; 23, Graney and Giesy 1986; 24, Graney and Giesy 1987; 25, Gupta and Durve 1984; 26, Kaila and Saarikoski 1977; 27, McKim et al. 1986; 28, Chapman and Shumway 1978; 29, Dominguez and Chapman 1984; 30, Nagler et al. 1986; 31, Lech et al. 1978; 32, Johnson and Finley 1980; 33, Mayer and Ellersieck 1986; 34, McKim et al. 1987; 35, Cote 1972; 36, Yousri and Hanke 1985; 37, Peer et al. 1983; 38 Johansen et al. 1987; 39, Mathers et al. 1985; 40, Brown et al. 1987; 41, Johansen et al. 1985; 42, Gupta 1983; 43, Pruitt et al. 1977; 44, Owen and Rosso 1981; 45, Huckins and Petty 1983; 46, Cleveland et al. 1982; 47, Parrish et al. 1978; 48, Trujillo et al. 1982; 49, Holmberg et al. 1972; 50, Tachikawa et al. 1987; 51, Borgmann and Ralph 1986; 52, Brown et al. 1985; 53, Norup 1972; 54, Holcombe et al. 1982; 55, Iwama and Greer 1979; 56, Webb and Brett 1973; 57, Niimi and McFadden 1982; 58, Glickman et al. 1977.

Table 5. Effects of pentachlorophenol on selected species of birds.

Species and other variables	Effect and reference
Mallard, Anas platyrhynchos	Acute oral LD-50 of 380 mg/kg body weight (BW); 95% confidence interval 205 to 704 mg/kg BW (Hudson et al. 1984).
Japanese quail, Coturnix japonica. Birds age 14 days were fed treated diets for 5 days, then untreated feed for 3 days.	No deaths at 3,100 mg/kg diet, 35% dead at 3,850, 50% at 5,139 (4,149 to 6,365), and 69% dead at 6,000 mg PCP/kg diet (Hill and Camardese 1986).
Domestic chicken, Gallus gallus. Fed diets containing 1, 10, 100, or 1,000 mg PCP/kg for 8 weeks.	Liver histopathology and diarrhea recorded in all treated groups vs. none in controls. The 1,000 mg/kg diet was the only ration to adversely affect the weight of all organs analyzed. After 5 weeks on PCP-free diet, residues were still measurable in adipose tissues of all treated birds (Stedman et al. 1980).
Domestic chicken. Fed dietary levels of 600, 1,200, or 2,400 mg/kg for 8 weeks.	No deaths in any group. Residues (mg/kg fresh weight) in 600 mg/kg group at 8 weeks were 2 in muscle, 10 in fat, 25 in liver and 80 in kidney vs. <0.02 in controls; no significant difference from controls in growth, blood chemistry, histopathology, or immune response. In the 1,200- and 2,400-mg/kg groups body weight decreased and liver weight increased; tissue residues were dose related (Prescott et al. 1982).
Domestic chicken. Raised over wood shavings containing 134 mg PCP/kg for 9 weeks.	The PCP residues (mg/kg FW) were 3.7 in liver 0.4 in muscle, and 0.3 in fat, vs. <0.08 in controls (Newsome et al. 1984).

Table 5. (Concluded)

Species and other variables	Effect and reference
Domestic chicken. Fed diets containing 10 mg PCP/kg for 14 days, then 21 days on PCP-free diets containing either no additives, 5% mineral oil, or 5% colestipol hydrochloride.	Body burden at 14 days was 362 ug/bird or 1.1 mg/kg BW. After 21 days, body burdens were 255 ug/bird in no-additive diet and nondetectable levels of <0.7 ug/bird in mineral oil and colestipol additive diets (Polin et al. 1986).
Domestic chicken. Eggs received single injected dose.	Hatching reduced 50% at a dose of 50 mg PCP/kg egg and 100% at 100 mg/kg (none hatched) (Stedman et al. 1980).
Ring-necked pheasant, Phasianus colchicus.	Acute oral LD-50 of 504 mg/kg BW; 95% confidence interval 343 to 743 mg/kg BW (Hudson et al. 1984).
Snail kite, Rostrhamus sociabilis. Found dead in rice fields after spraying to control populations of snails (Pomacea glauca, P. lineata)--the exclusive food of snail kites. Residues in soft parts of snails found dead after spraying were about 37 mg PCP/kg fresh weight.	Residues in dead snail kites, in mg PCP/kg FW, were 46 in liver, 20 in kidney, and 11 in brain vs. <0.2 in controls, and 2 to 17 in birds surviving exposure (Vermeer et al. 1974).
Canary, Serinus canarius. Nesting on straw containing 285 mg PCP/kg.	Reduced hatch, high mortality of young during the first week posthatch, and none surviving to age 3 months. Prior to death, young showed inhibited growth and feather development. Adult birds appeared normal (Dorrestein and Zella 1979).

and liver and lower in other tissues; the high residues may reflect the principal routes of metabolism and excretion (Stedman et al. 1980). The loss of body fat in chickens, accomplished by feeding bile acid binding resins, hastens PCP excretion (Table 5; Polin et al. 1986).

Spraying of PCP to control populations of water snails in rice fields of Surinam resulted in the death of fish and birds, including snail kites (Rostrhamus sociabilis), certain eqrets and herons, and wattled jacanas (Jacana jacana). Levels of PCP in these birds and their food items suggested that PCP-contaminated food probably caused the deaths (Vermeer et al. 1974; Table 5).

Pentachlorophenol is widely used as a wood preservative, which often results in residues in wood shavings used as poultry litter. A moldy smell and taste in chicken tissue has been traced to the presence of chloroanisoles formed from PCP and tetrachlorophenol in the bedding. Several dioxins, diphenyl ethers, dibenzofurans, and 2-phenoxyphenols have also been identified (Newsome et al. 1984). For example, PCP-contaminated (134 mg/kg) commercial wood shavings used as chicken litter contained detectable levels of heptachlorinated diphenyl ethers (18 ug/kg), octachlorinated diphenyl ethers (12 ug/kg), nonachlorinated diphenyl ethers (6 ug/kg), octachlorinated 2-phenoxyphenols (299 ug/kg), nonachlorinated 2-phenoxyphenols (50 ug/kg), heptachlorinated dibenzodioxins (19 ug/kg), and octachlorinated dibenzodioxins (143 ug/kg). After 9 weeks, PCP was detectable in liver, fat, and muscle; chlorinated diphenyl ethers were detectable in fat, but not in muscle or liver; octa- and nonachlorinated 2-phenoxyphenols were found in all three tissues; and dioxins only in liver and fat (Newsome et al. 1984). Exposure of domestic chickens to litter contaminated with PCP enhanced susceptibility to common poultry pathogens, perhaps due to immunosuppresion by the chemical (Prescott et al. 1982).

MAMMALS

Data are scarce on the biological effects of PCP on mammalian wildlife, although evidence continues to accumulate on this subject for man, livestock, and small laboratory animals. Available data on PCP and mammals are briefly summarized, but it is not now clear if these findings are applicable to representative species of sensitive mammalian wildlife.

Pentachlorophenol tends to accumulate in mammalian tissues unless it is efficiently conjugated into a readily excretable form (Kinzell et al. 1985). The ability to conjugate PCP varies widely among species (Braun and Sauerhoff 1976; EPA 1980). For example, both laboratory rats (Rattus sp.) and humans eliminate about 75% of all PCP in the urine in an unconjugated form, but rhesus monkeys (Macaca mulatta) are unable to excrete PCP efficiently, whereas

mice were the most efficient. As one result, $Tb\frac{1}{2}$ values were low (about 24 hours) for mice, high (up to 360 hours) for rhesus monkey, and intermediate for rats and humans (EPA 1980). In man, however, the observed elimination half-life indicates that steady-state body burdens are 10 to 20 times higher than values extrapolated from animal pharmacokinetic data (Uhl et al. 1986).

Pentachlorophenol is not a carcinogen, and the evidence for mutagenicity is mixed. No carcinomas were produced in rodents, regardless of the composition of the PCP solution tested or route of exposure (EPA 1980; Choudhury et al. 1986). Some studies suggested that PCP may be mutagenic in the bacterium Bacillus subtilis, the yeast Saccharomyces cerevisiae, and in laboratory mice (Mus sp.), but not in two other species of bacteria tested--Salmonella typhimurium and Escherichia coli (Choudhury et al. 1986).

The primary sources of PCP in man include direct intake by way of diet, air, or water and through contact with PCP-contaminated materials (Uhl et al. 1986). The chief routes of exposure in an industrial setting are by way of inhalation and skin contact. Percutaneous absorption is significantly enhanced when PCP is dissolved in organic solvents, such as fuel oil, or when PCP comes in contact with open cuts and scratches (Wood et al. 1983). Pentachlorophenol has resulted in death in man through suicide and occupational and accidental exposures (EPA 1980; Rozman et al. 1982; Lambert et al. 1986).

Cases of PCP poisoning, including fatalities, were characterized by high fever, renal insufficiency, profuse perspiration, rapid heart beat and breathing, abdominal pain, dizziness, nausea, spasms, and death 3 to 25 hours after onset of symptoms (Knudsen et al. 1974; EPA 1980; Wood et al. 1983). Postmortem examination showed kidney degeneration, inflamed gastric mucosa, edematous lungs, and centrilobular degeneration of liver (EPA 1980; St. Omer and Gadusek 1987). Symptoms of nonfatal PCP intoxication in man include conjunctivitis, chronic sinusitus, nasal irritation, upper respiratory complaints, sneezing, coughing, recurring headache, neurological complaints, weakness, and several types of skin lesions (Knudsen et al. 1974; EPA 1980; Rozman et al. 1982). All symptoms were related to proximity to PCP-treated wood, and sometimes to elevated PCP residues in serum and urine (Lambert et al. 1986). At the cellular level, PCP--like other halogenated phenols--uncouples oxidative phosphorylation. A possible antidote to PCP poisoning is the administration of cholestyrmine, a compound that interferes with the enterohepatic cycle of PCP, and also increases its elimination directly across the intestinal wall (Rozman et al. 1982).

The exposure of livestock to PCP can result from ingestion of feeds stored or fed in PCP-treated wooden structures, licking of treated wood, cutaneous absorption by direct contact with treated wood, and inhalation of air containing preservative chemicals--particularly volatile chlorophenols (Forsell et al. 1981). Acute signs of PCP intoxication in various domestic and laboratory animals include elevated blood sugar, vomiting, elevated blood

pressure, increased respiration rate, tachycardia, motor weakness, weakened eye reflex, frequent defecation, high fever, collapse, asphyxial convulsions, and death followed by rapid rigor mortis (Knudsen et al. 1974; Nishimura 1984; St. Omer and Gadusek 1987). In domestic cattle (Bos sp.) PCP has also been associated with decreased milk production, skin lesions, increased mastitis, persistent infections, and reduced survival (Forsell et al. 1981).

Among sensitive species of mammals tested against PCP (Table 6), acute oral LD-50 values ranged from 27 to 300 mg/kg BW, but most values were between 55 and 150 mg/kg BW. Sublethal effects were noted at much lower concentrations than those causing death. They included elevated tissue residues at dietary intakes equivalent to 0.05 mg/kg BW, or atmospheric concentrations >0.1 mg/m^3; organ damage at 0.2 to 2.0 mg/kg BW; reproductive impairment at >1.25 mg/kg BW; and retarded growth and reproduction in animals fed rations containing >30 mg/kg (Table 6).

Many commercial lots of technical PCP are known to contain small--but possibly biologically significant--amounts of highly toxic dioxins, dibenzofurans, and hexachlorobenzene. These contaminants may be responsible for most of the toxicity of technical PCP preparations (McConnell et al. 1980; Parker et al. 1980; Wollesen et al. 1986; Holsapple et al. 1987). However, both technical and analytical grade PCP can induce hepatic mixed function oxidases in intoxicated rats and cattle. In cattle, this effect was observed in both calves and adults, and in hepatic as well as pulmonary microsomes, and seemed to be dose related (Shull et al. 1986).

Table 6. Effects of pentachlorophenol on selected mammals.

Organism, dose, and other variables	Effects and reference
Domestic cattle, Bos taurus	
Oral dose of 0.05 or 0.5 mg/kg BW.	Maximal plasma values of 1.5 and 9.6 mg/l, respectively. Calves fed grain and hay from a PCP-treated feeder for 10 days contained plasma PCP levels of 1.1 mg/l, but levels returned to normal after access to feeder was denied (Osweiler et al. 1984).
Fed equivalent of 0.2 mg/kg BW daily for 75 to 84 days, then 2.0 mg/kg BW daily for 50 to 62 days.	No effect on milk production, feed intake, body weight, lymphocyte function, or histopathology of spleen, thymus, or lymph nodes. Postmortem examination showed enlarged liver, lungs, kidneys, and adrenals; significant loss of renal function (Forsell et al. 1981; Kinzell et al. 1981).
Fed 0.2 mg/kg BW daily for 95 days, then given single oral dose of uniformly ring-labeled C^{14}-PCP; analyzed 4 days postadministration.	Highest residues were in liver, kidney, and lungs; in milk, the fat fraction contained the greatest amount. $Tb\frac{1}{2}$ for absorption was 4.3 hours, and for elimination 43 hours. Most excretion was by way of urine (76%), then milk, and feces (5% each). In urine, PCP was present in the conjugated form (Kinzell et al. 1985).
Fed 20 mg technical grade PCP/kg BW daily for 10 days, then 10 mg/kg BW for an additional 60 days.	No clinical effects noted during the 70-day treatment or during a 165-day posttreatment period. Contaminants in PCP--including several dioxins and hexachlorobenzene--were found in milk, fat, and blood. PCP residues in whole milk rose to 4 mg/kg, but declined to <0.1 mg/kg within a few days after PCP cessation (Firestone et al. 1979).

Table 6. (Continued)

Organism, dose, and other variables	Effects and reference
Female yearling Holsteins were fed technical or analytical grade PCP in diets at 20 mg/kg BW daily for 42 days (647 mg/kg diet), then 15 mg/kg BW daily for 118 days (491 mg/kg diet).	Technical grade PCP was related to decreased body weight and feed conversion efficiency, anemia, enlarged liver and lungs, decreased thymus weight, and lesions in urinary bladder mucosa. Holsteins exposed to analytical grade PCP were comparable to controls. The toxicity of PCP in cattle seems to be due to its contamination with toxic impurities, especially dioxins (McConnell et al. 1980; Parker et al. 1980).
140 mg/kg BW.	Acute oral LD-50 (Knudsen et al. 1974).
Domestic dog, Canis familiaris	
150 to 200 mg/kg BW.	Acute oral LD-50 (Knudsen et al. 1974).
Guinea pig, Cavia porcellus	
100 mg/kg BW.	Acute oral LD-50 (Choudhury et al. 1986).
Hamster, Cricetus spp.	
1.25 to 20 mg/kg BW.	Fetal deaths and resorption (EPA 1980).
Man, Homo sapiens	
Average concentrations of 0.0012 to 0.18 mg/m^3 air for 3 to 34 years among occupationally exposed workers.	Concentrations of PCP in blood of 20 workers ranged between 0.023 and 0.775 mg/l and were below the "biological tolerance value" of 1.0 mg/l; no effect on sister chromatid exchange or chromosomal aberrations (Ziemsen et al. 1987).

Table 6. (Continued)

Organism, dose, and other variables	Effects and reference
< 0.02 to > 0.1 mg/l urine in occupationally exposed woodworkers	During a 16-day vacation and plant shutdown, urinary excretion accounted for 90% elimination in those with high initial PCP levels (i.e., >0.1 mg/l) to 67% elimination in those with initial urinary levels of 0.02 to 0.1 mg PCP/l, and to 34% reduction in workers with <0.02 mg/l; $Tb\frac{1}{2}$ was estimated at 33 hours in urine and 30 hours in plasma (Kalman and Horstman 1983).
Single oral dose of 0.1 mg/kg BW.	In urine of four subjects, 74% was eliminated unchanged and 12% as PCP-glucuronide; 4% was eliminated in feces as PCP and PCP-glucuronide. A peak blood level of 0.25 mg/l was reached 4 hours after dosing (EPA 1980).
>1.0 mg/m^3 air.	Painful irritation in upper respiratory tract, sneezing, and coughing in persons newly exposed to PCP; up to 2.4 mg/m^3 can be tolerated by conditioned individuals (EPA 1980).
Total dose of 3.9 to 18.0 mg.	Steady state attained in about 3 months; $Tb\frac{1}{2}$ values of 20 days in whole body and about 17 days in urine and blood (Uhl et al. 1986).
8 to 1,130 mg/kg fresh weight tissue.	Residues in tissues of 33-year-old male who died after working in chemical plant for 3 weeks; job involved breaking up large blocks of PCP with jackhammer. Before death, high body temperature and coma. After death, rigor mortis was profound and immediate. Postmortem examination showed cerebral edema, fatty degeneration of viscera, and PCP levels (mg/kg) of 8 in stomach contents, 29 in urine, 52 in liver, 116 in lung, 162 in blood, 639 in kidney, and 1,130 in bile (Wood et al. 1983; Gray et al. 1985).

Table 6. (Continued)

Organism, dose, and other variables	Effects and reference
28 to 225 mg/kg fresh weight tissue.	Residues associated with acute toxicosis and death were 28 to 123 in kidney, 50 to 176 in blood, and 62 to 225 in liver (EPA 1980).
75 to 225 mg/kg fresh weight tissue.	Residues in tissues of PCP suicide victim were 75 in urine, 116 in kidney, 173 in blood, and 225 in liver (EPA 1980).
4,000 mg/l solution.	Immersion of hands for 10 minutes produced skin irritation (EPA 1980).
Domestic cat, Felis domesticus	
Pine wood shavings used as litter, containing 470 mg PCP/kg.	Of 14 cats in contact with litter, 3 died and 8 became ill but recovered. Maximum PCP residues (mg/kg) in the dead cats were 20 in liver, 24 in kidney, and 10 in stomach contents (Peet et al. 1977).
Rhesus monkey, Macaca mulatta	
10 mg/kg BW, single oral dose.	Residues highest in liver and GI tract; all other tissues contributed <4% of total body burden (EPA 1980).
10 mg/kg BW, single oral dose.	$Tb\frac{1}{2}$ values in blood and urine were 41 to 72 hours in males and 84 to 92 hours in females (Braun and Sauerhoff 1976).
50 mg/kg BW, repeated 4 weeks later.	During the first day after each dose, 20% was excreted into urine 0.5% into feces, and 20% into bile. The addition of 4% cholestyramine to diets for 6 days resulted in increased fecal excretion by a factor of 18X and increased total body burden excretion by 1.4X (Rozman et al. 1982).

Table 6. (Continued)

Organism, dose, and other variables	Effects and reference
Mouse, Mus musculus	
Fed diets equivalent to 3 mg/kg BW daily for 24 months.	No measurable effect in females, based on clinical chemistry, hematology, routine histopathology, and organ weight changes (EPA 1980).
Fed diets equivalent to 10 mg/kg BW daily for 22 months.	No measurable effect in males, based on clinical chemistry, hematology, routine histopathology, and organ weight changes (EPA 1980).
15 to 37 mg/kg BW through intraperitoneal or subcutaneous route	$Tb\frac{1}{2}$ of about 24 hours through urinary excretion (EPA 1980).
Fed diets containing 50 mg pure (99%) PCP/kg or technical grade (86%) for 10 to 12 weeks.	Mice exposed to technical grade PCP had enhanced tumor susceptibility (1.9X) from transplanted tumors; mortality increased 2.4X over controls after sarcoma virus inoculation. Mice exposed to pure PCP showed no enhanced growth of reduced tumors, but developed splenic tumors: 22% vs. none in controls (Kerkvliet et al. 1982).
65 to 252 mg/kg BW.	Acute oral LD-50; females more sensitive than males (Knudsen et al. 1974; Borzelleca et al. 1985; Choudhury et al. 1986).
White rabbit, Oryctolagus cuniculus	
1 to 3 mg/kg BW daily for 90 days administered orally.	No signs of intoxication (EPA 1980).
39 mg/kg BW.	Lethal cutaneous dose administered in pine oil (Cote 1972).

Table 6. (Continued)

Organism, dose, and other variables	Effects and reference
60 to 200 mg/kg BW.	Acute dermal lethal dose (EPA 1980).
100 to 130 mg/kg BW.	Acute oral LD-50 (Knudsen et al. 1974; Choudhury et al. 1986).
350 mg/kg BW.	Lethal cutaneous dose administered in olive oil (Cote 1972).
Domestic sheep, Ovis aries	
120 mg/kg BW.	Acute oral LD-50 (Knudsen et al. 1974).
Pipistrelle bat, Pipistrellus pipestrellus	
Females roosting in contact with timbers treated with 5% PCP solutions.	All bats introduced 6 weeks postapplication died in 3 to 7 days; those introduced 8 weeks postapplication died in 1 to 2 days; bats in contact with timbers 14 months postapplication died in 5 to 23 days (Racey and Swift 1986).
Laboratory rat, Rattus norvegicus	
Dietary levels of 20, 100, and 500 mg/kg feed equivalent to 1.2, 6, and 30 mg/kg BW daily, respectively.	No effect after exposure for 8 months to pure grade PCP at doses of 20 and 100 mg/kg. Technical grade PCP produced disruptions in liver enzyme activity in females at 20 and 100 mg/kg. At 500 mg/kg, body weight gain was reduced in both sexes and by both grades of PCP (EPA 1980).
Diets containing 25, 50, or 200 mg PCP/kg, equivalent to 1.5, 3, and 12 mg/kg BW daily, respectively.	After 12 weeks, no observable effect at 25 mg/kg diet; dose-related adverse effects on liver, kidney calcium deposits, and blood chemistry at higher dietary levels (Knudsen et al. 1974).

Table 6. (Continued)

Organism, dose, and other variables	Effects and reference
Dietary level of 50 mg/kg, equivalent to about 3 mg/kg BW daily.	After 62 days, no observable effect on reproduction, neonatal growth, survival, or development. After 12 weeks, males had decreased hemoglobin and erythrocytes. After 2 years, no significant adverse effects on growth, survival, reproduction, or development (Schwetz et al. 1978; EPA 1980; McConnell et al. 1980).
5.0 to 5.8 mg/kg BW on days 6 to 15 of gestation.	Delayed ossification of skull (EPA 1980).
10 mg/kg BW.	After a single oral dose, 0.44% remained after 9 days; 82% of total residue was in kidney and liver, and lowest residues were in brain, spleen, and fat. A maximum residue of 45 mg/l was attained in blood plasma; the $Tb\frac{1}{2}$ in plasma was 13 to 121 hours (EPA 1980).
10 or 100 mg/kg BW.	After a single dose, elimination occurred by way of several routes: catabolism to tetrachlorohydroquinone; excretion of unchanged PCP and its glucuronide conjugate in urine; excretion of PCP or its metabolites into bile. More than 90% was eliminated during the rapid phase, the $Tb\frac{1}{2}$ being 13 to 17 hours (Braun et al. 1977).
Dietary levels of 200 mg/kg, equivalent to about 13 mg/kg BW daily.	After 181 days, reduction in crown-rump length and increase in fetal skeletal variations (Welsh et al. 1987).
15 mg/kg BW daily of purified PCP on days 6 to 15 of gestation.	No measurable effect on development (EPA 1980).
27 to 300 mg/kg BW.	Acute oral LD-50 range. Lower values in tests when PCP dissolved in fuel oil, in weanling rats, and in adult rats; higher values in tests with juveniles and when immersion vehicle is peanut oil (Knudsen et al. 1974; McConnell et al. 1980; Borzelleca et al. 1985; Choudhury et al. 1986; St. Omer and Gadusek 1987).

Table 6. (Continued)

Organism, dose, and other variables	Effects and reference
Dietary levels equivalent to 30 mg/kg BW daily.	Decrease in neonatal survival and growth after 62 days. After 2 years, no evidence of carcinogenicity but adverse effects on adult growth and serum enzyme activity levels (Schwetz et al. 1978).
50 mg/kg BW on days 6 to 15 of gestation.	100% fetal resorption (EPA 1980).
60 mg/kg BW.	Single dose on day 9 or 10 of gestation produced reduction in fetal weight; no effect when given on days 11, 12, or 13 (EPA 1980).
120 mg/kg BW.	Single dose results in early hepatic glycogen depletion and elevation in blood glucose (Nishimura 1984).
Domestic pig, Sus spp.	
5, 10, or 15 mg purified PCP/kg BW daily for 30 days.	No effect on weight gain, food consumption, or kidney weight. Significantly enlarged liver in 10 and 15 mg/kg groups. Elevated PCP residues in all groups. Residues in 5 mg/kg group vs. controls, in mg/kg fresh weight, were: 78.1 vs. 0.7 for blood, 6.7 vs. 0.2 for muscle, 22.0 vs. 0.2 for kidney, and 28.9 vs. 0.5 for liver (Greichus et al. 1979).
27 to 55 mg/kg BW.	Fatal chronic dose (Schipper 1961).
30 mg/kg BW daily for 7 days.	Acute toxicosis (Greichus et al. 1979).
Pregnant swine in direct contact with lumber freshly treated with PCP.	Extensive mortality in newborn swine (Schipper 1961).

Table 6. (Concluded)

Organism, dose, and other variables	Effects and reference
Eastern chipmunk, *Tamias striatus*	
138 mg/kg BW.	Acute oral LD-50 (Ege 1985).
200 mg/kg BW.	Acute oral LD-100 (Ege 1985).
Fed diets containing 250 or 500 mg PCP/kg for 2 weeks.	No increase in metabolic activity. Some weight loss due to food avoidance; enlarged livers (Ege 1985).

CURRENT RECOMMENDATIONS

Commercial PCP preparations often contain variable amounts of chlorophenols, hexachlorobenzene, phenoxyphenols, dioxins, dibenzofurans, chlorinated diphenyl ethers, dihydroxybiphenyls, anisoles, catechols, and other chlorinated dibenzodioxin and dibenzofuran isomers. These contaminants contribute to the toxicity of PCP, sometimes significantly, although the full extent of their interactions with PCP and with each other in PCP formulations are unknown. Unless these contaminants are removed or sharply reduced in existing technical and commercial grade PCP formulations, efforts to establish sound PCP criteria for protection of natural resources may be hindered.

Proposed PCP ambient water quality criteria to protect freshwater and marine life now range from 48 to 55 ug/l for acute effects, 3.2 to 34 ug/l for chronic effects, and daily mean concentrations of 6.2 ug/l, not to exceed 140 ug/l (Table 7). Available data, however, suggest that significant adverse effects occur at much lower PCP concentrations--i.e., between 0.035 and 19 ug/l. In rainbow trout (Salmo gairdneri), for example, concentrations of 0.035 to 1.0 ug/l produced elevated tissue residues (Choudhury et al. 1986; McKim et al. 1986), 7.4 ug/l caused growth inhibition in 28 days (EPA 1980), and 10 to 19 ug/l produced adverse effects and some deaths (Chapman and Shumway 1978; Dominguez and Chapman 1984). Other sensitive fish species include sockeye salmon (Oncorhyncus nerka), showing growth inhibition after prolonged exposure to 1.8 or 3.2 ug PCP/l (EPA 1980; Choudhury et al. 1986); larvae of common carp (Cyprinus carpio), having an 96-hour LC-50 of 9.5 ug/l (Choudhury et al. 1986); and largemouth bass (Micropterus salmoides), exhibiting reduced food conversion efficiency at 10 ug/l (Johansen et al. 1987). Among sensitive species of plants and invertebrates, American oysters (Crassostrea virginica) have elevated tissue residues after exposure for 28 days to 2.5 ug/l (Schimmel et al. 1978); cladocerans have impaired reproduction at 4.1 ug/l (Hedtke et al. 1986); alga show chlorosis inhibition in 72 hours at 7.5 ug/l (EPA 1980); and estuarine macrobenthos populations decreased in abundance and species after exposure for 13 weeks to 15.8 ug/l (Tagatz et al. 1981). Also, an air concentration of 0.2 mg/m^3--a tolerable level to humans (Table 7)--interfered with photosynthesis in duckweed, Lemna minor (Huber et al. 1982).

As judged by these studies, it seems appropriate to suggest modification of certain aquatic PCP criteria. A maximum PCP concentration of 3.2 ug/l is

Table 7. Proposed pentachlorophenol criteria for protection of fish, wildlife, and humans.

Resource and criterion	Concentration or dose	Reference[a]
AQUATIC BIOTA		
Freshwater life		
Acute	48 to <55 ug/l	1,2,3
Chronic	<3.2 ug/l	1
24-hour mean	<6.2 ug/l	2
Maximum concentration	<140 ug/l	2
Fish		
Warmwater species	10 to <15 ug/l	4
Coldwater species	20 to <40 ug/l	4
Saltwater life		
Acute	<53 ug/l	1
Chronic	<34 ug/l	1,5
BIRDS		
Tissue residues		
Contaminated	>2 mg/kg fresh weight	6
Life-threatening	>11 mg/kg fresh weight	6
Diets		
Adverse effects	>1.0 mg/kg diet	7
Fatal	>3,850 mg/kg diet	8
Wood shavings		
Litter	<134 mg/kg	9
Nesting materials	<285 mg/kg	10
LIVESTOCK AND LABORATORY MAMMALS		
No measurable adverse effects		
Rat		
Females	3 mg/kg BW daily for 24 months	1,11
Males	10 mg/kg BW daily for 22 months	1,11
Rabbit	3 mg/kg BW daily for 90 days	1

Table 7. (Continued)

Resource and criterion	Concentration or dose	Reference[a]
Adverse effects		
Elevated tissue residues, cattle		
Blood plasma	0.05 mg/kg BW, single dose	12
Internal organs	0.2 mg/kg BW for 95 days	13
Histopathology, cattle		
Internal organs	0.2 mg/kg BW daily for about 80 days, then 2.0 mg/kg BW daily for about 59 days	14,15
Internal organs	50 mg/kg diet for 12 weeks, equivalent to 3 mg/kg BW daily	16
Reproductive impairment		
Hamster	1.25 to 20 mg/kg BW, single dose	1
Rat	5 mg/kg BW daily or 50 mg/kg diet daily, chronic	5
Rat	5 to 5.8 mg/kg BW daily	1,17
Increased tumor frequency		
Mice	50 mg/kg diet, chronic	18
Death, various species		
Acute oral LD-50	55 to 200 mg/kg BW	5,16,19,20
Acute dermal LD-50	60 to 200 mg/kg BW	1
Contaminated wood shavings, dermal contact	470 mg/kg	21
HUMAN HEALTH		
Current exposure levels, 70 kg adult		
Food	15 ug daily, or 0.21 ug/kg BW daily	1
Water	0.12 ug daily, or 1.7 ug/kg BW daily	1
No adverse effect levels		
Food, upper safe limit, 70 kg adult	30 ug/kg BW, or 2.1 mg per person	1
Wood, in contact with food	Up to 50 mg/kg	1
Drinking water	21 ug/l[b]	1,5,22
Upper safe limit	1.01 mg/l	1

Table 7. (Continued)

Resource and criterion	Concentration or dose	Reference[a]
Air, 8 hours exposure daily, 5 days weekly	<0.5 mg/m^3	1,5,23,24
Blood	<1.0 mg/l	25
Blood plasma	<0.5 mg/l	24
Total intake	<3 ug/kg BW daily[c]	22
Total intake, 70 kg adult	<30 ug/kg BW daily, or 2.1 mg daily[d]	5
Expectant mothers	No safe level established to guard against fetal toxicity	17
Adverse effect levels		
Air	>1.0 mg/m^3	1
Dermal solutions	4,000 mg/l	1
Tissue residues associated with acute toxicosis		
Kidney	>28 mg/kg fresh weight	1
Blood	>40 to 80 mg/l	1,23
Liver	>62 mg/kg fresh weight	1
Tissue residues associated with death		
Stomach contents	8 mg/kg fresh weight	24,26
Urine	29 to 75 mg/l	1,24,26
Liver	52 to 225 mg/kg fresh weight	1,24,26
Lung	116 mg/kg fresh weight	24,26
Blood	162 to 176 mg/l	1,24,26
Kidney	116 to 639 mg/kg fresh weight	1,24,26
Bile	1,130 mg/kg fresh weight	24,26

Table 7. (Concluded)

[a]References: 1, EPA 1980; 2, Zischke et al. 1985; 3, Johansen et al. 1987; 4, Hodson and Blunt 1981; 5, Choudhury et al. 1986; 6, Vermeer et al. 1974; 7, Stedman et al. 1980; 8, Hill and Camardese 1986; 9, Newsome et al. 1974; 10, Dorrestein and Zelle 1979; 11, Schwetz et al. 1978; 12, Osweiler et al. 1984; 13, Kinzell et al. 1985; 14, Forsell et al. 1981; 15, Kinzell et al. 1981; 16, Knudsen et al. 1974; 17, Williams 1982; 18, Kerkvliet et al. 1982; 19, Schlipper 1961; 20, Ege 1985; 21, Peet et al. 1977; 22, Lu et al. 1978; 23, Cote 1972; 24, Wood et al. 1983; 25, Ziemsen et al. 1987; 26, Gray et al. 1985.

[b]Based on no observable adverse effect level of 3 mg/kg BW daily in rat study, uncertainty factor of 1,000X, and water consumption of 2 liters daily.

[c]Based on animal data and uncertainty factor of 1,000X.

[d]Based on rat chronic oral no observable effect level of 3 mg/kg BW daily and uncertainty factor of 100.

indicated, and this level would probably protect most aquatic species, although it would not prevent accumulations and growth inhibition in salmonids or accumulations in oysters. Additional research is needed to establish sound water quality criteria for PCP, and also to interpret the significance of its residues and their metabolites in tissues of representative species.

Dietary concentrations of 1.0 mg/kg and higher produced diarrhea and liver histopathology in chickens (Gallus spp.) after 8 weeks (Stedman et al. 1980), and deaths occurred at relatively high dietary concentrations, i.e., 3,850 mg/kg, in Japanese quail (Coturnix japonica) after 5 days (Hill and Camardese 1986). Wood shavings contaminated with PCP produced elevated residues when used as litter for domestic chickens (Newsome et al. 1984), and death in canaries (Serinus canarius), when used as nesting materials (Dorrestein and Zelle 1979). Tissue residues >2 mg/kg fresh weight are considered to be indicative of significant environmental PCP contamination, and those >11 mg/kg fresh weight were associated with birds that died or were recovering from PCP exposure (Table 7; Vermeer et al. 1974). No data are now available on avian wildlife and PCP contamination in their diets, residues in their tissues, or frequency of use of PCP-contaminated wood shavings for nesting materials and other purposes.

As judged by studies with domestic and small laboratory mammals, no observable adverse effects have been noted at dietary levels equivalent to 3 to 10 mg PCP/kg BW (Table 7). Variability is great among species, however, and adverse effects have been documented in some species (Table 7) at doses as low as 0.05 to 0.2 mg/kg BW (elevated tissue residues), 0.2 to 2.0 mg/kg BW or 50 mg/kg diet (histopathology, reproductive impairment, increased tumor frequency), and 55 to 60 mg/kg BW (death). Based on guidelines for carcinogen risk assessment and inadequate evidence for animal carcinogenicity or absence of human cancer data, PCP is classified as group D, meaning that it is not classified as a human carcinogen (Choudhury et al. 1986).

Data for man indicate that adverse effects occur at concentrations in air >1.0 mg PCP/m^3 and in tissues >8 mg/kg fresh weight (Table 7). No adverse effects were noted at daily intakes of 2.1 mg per 70-kg adult or 30 ug/kg BW, up to 1.01 mg/l in drinking water, <0.5 mg/m^3 in air, <0.5 mg/l in blood plasma, and <1.0 mg/l in blood (Table 7). It is noteworthy that the currently recommended PCP air concentration of 0.5 mg/m^3 results in a daily intake of 2.5 to 3.8 mg (based on 15 to 23 m^3 of air inhaled daily, 8 hour exposure), equivalent to 42 to 63 ug/kg BW for a 60 kg female. These levels are higher than the currently recommended no adverse effect level of 30 ug/kg BW daily (Table 7), and overlap or exceed the 58 to 74 ug/kg BW daily range--a level recommended by Williams (1982). Air concentrations >1.0 mg PCP/m^3 can produce respiratory irritation in unacclimatized individuals, but concentrations as high as 2.4 mg/m^3 can be tolerated by conditioned individuals (EPA 1980). The biological tolerance value of <1,000 ug PCP/l in blood, recommended by Ziemsen et al. (1987), is based on occupational air exposure studies: exposure to maximum average air concentrations of 0.18 mg PCP/m^3 for up to 34 years

produced blood PCP residues of 23 to 775 ug/l, with no measurable adverse effects. The authors concluded that PCP and its impurities in occupationally relevant concentrations below the maximum concentration in the workplace and below the biological tolerance value do not produce genotoxic damage that can be detected on the chromosomal level, either in vivo or in vitro.

The human taste threshold for PCP in drinking water is about 30 ug/l (EPA 1980), a level far below the upper safe limit of 1.01 mg/l and near the no observable effect level of 21 ug/l (Table 7). Odor detection is not as sensitive as taste: the odor threshold for PCP ranges from about 857 ug/l at 30 ^{0}C, to 1,600 ug/l at 20 to 22 ^{0}C, to 12,000 ug/l at 60 ^{0}C (EPA 1980). It is not clear whether the determined organoleptic threshold values made the water undesirable or unfit for consumption (EPA 1980). If fish and wildlife species of concern have PCP organoleptic thresholds that are similar to those of humans, or lower, will they too avoid contaminated habitats or diets?

Data for PCP and terrestrial wildlife are incomplete and--in view of the large interspecies variations in sensitivity--need to be collected. Until they are, it seems reasonable to apply to wildlife the same levels recommended for human health protection.

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Pentachlorophenol (PCP) is now widely used as a wood preservative; this use has contributed to the presence and detection of PCP residues in air, rain, groundwater, surface water, fish and aquatic invertebrates, and human urine, blood, and milk. This report reviews the technical literature on ecological and toxicological aspects of PCP in the environment, with emphasis on fishery and wildlife resources. Subtopics include sources and uses, chemical properties, fate, background concentrations, lethal and sublethal effects, and current recommendations for resource protection.

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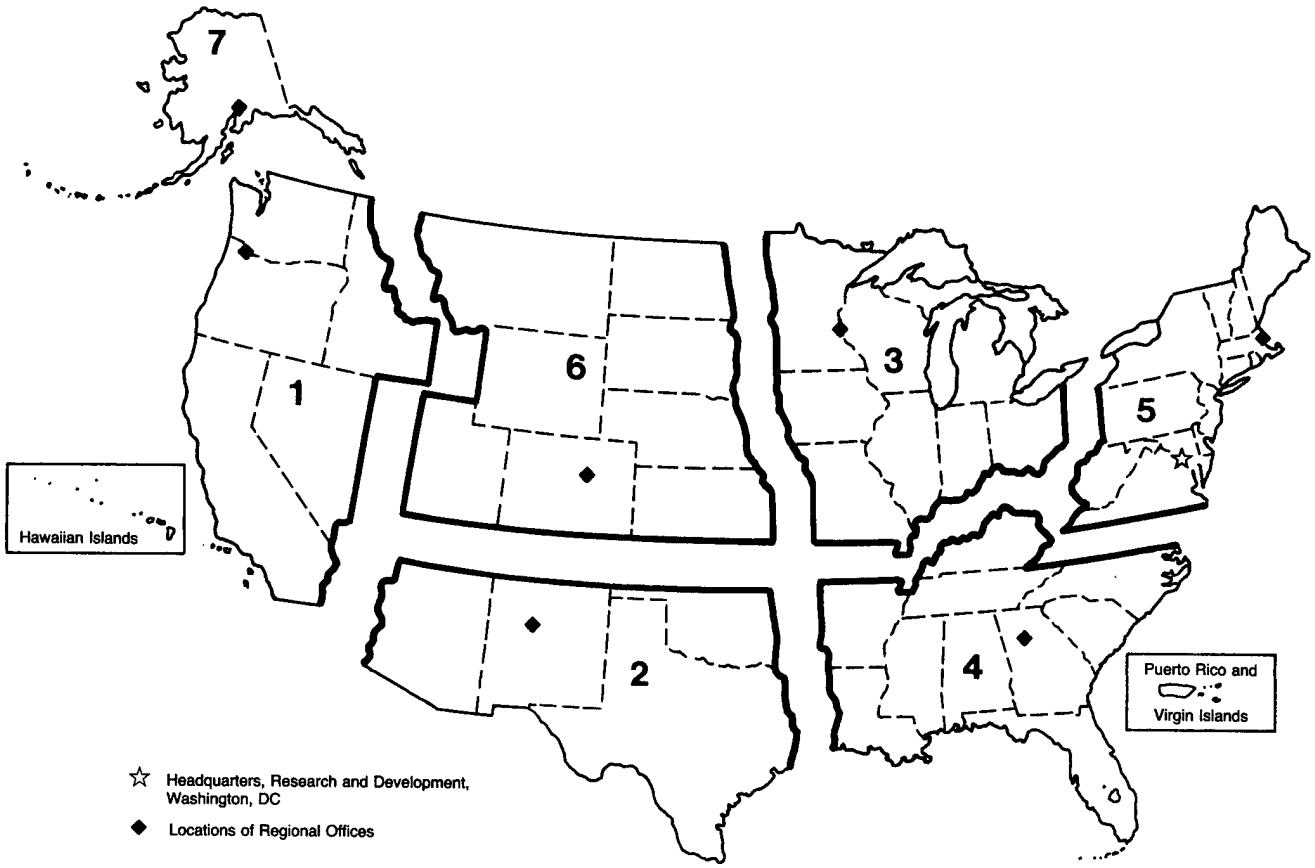

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